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THE CANADIAN ORTHOPAEDIC ASSOCIATION: A HISTORICAL REVIEW

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For many years before the Canadian Orthopaedic Association was founded, orthopedic surgery was practised in various centres in Canada by men who can be called the fathers of Canadian orthopedic surgery. No account of the Association would be complete without some mention of them for they were our preceptors and some were even the preceptors of our teachers. They deserve our admiration for their dedication to their specialty at a time when the maintenance of its standards was a difficult and lonely task. To a considerable degree they practised in isolation. In this country of great distances they had few opportunities to share their problems and discuss their achievements with fellow Canadians or to develop a distinctive Canadian school of orthopedic surgery. Of necessity they turned to the United States, to England and to Europe for the contacts that kept them abreast of progress in this rapidly expanding field. They command our respect for the work they did in founding the specialty of orthopedic surgery in Canada.

The first World War did much to draw Canadian orthopedic surgeons together through the necessity of treating war casualties in army centres overseas and later in veterans' hospitals in this country, but it was not until the Canadian Orthopaedic Association was founded that we began to develop a truly Canadian orientation in orthopedic surgery.

It was in 1943 that the initial steps were taken. The initiative came from the Montreal Orthopaedic Society led by its President, J. Edouard Samson, and by J. Appleton Nutter who had founded that association in 1934. In October 1943 the Society called a meeting to discuss the possibility of founding a Canadian orthopedic association. To this meeting they invited Dr. Andrew P. MacKinnon of Winnipeg and Dr. Robert I. Harris of Toronto. A committee was formed, composed of all the past presidents of the Montreal Orthopaedic Society. The committee was asked to draft plans for the project. After the presentation of its report to a meeting of the Montreal Orthopaedic Society on February 19, 1944, a Provisional Founding Committee was appointed and charged with the responsibility of founding a Canadian orthopedic association. It was also to act as the Provisional Executive Committee until the association was legally constituted. The personnel of this committee were: President—Dr. J. Appleton Nutter; Vice-Presidents—Drs. Robert I. Harris, Andrew P. MacKinnon and J. Edouard Samson; and General Secretary—Dr. J. Calixte Favreau.

On May 24, 1944, this committee met in Toronto during the meeting of The Canadian Medical Association. A Committee on By-Laws was appointed under the chairmanship of Dr. Samson and other business was conducted. Later in the day a number of Canadian orthopedic surgeons met with the Committee to discuss progress. With minor revisions they approved the activities of the Committee to that date. During this period of organization all orthopedic surgeons in Canada, as well as those Canadians who were overseas at that time, were informed of the proceedings of the Provisional Executive Committee.

It was planned that the first annual meeting of the newly organized Canadian Orthopaedic Association would be held at the Mount Royal Hotel, Montreal, on June 11 and 12, 1945, by courtesy of The Canadian Medical Association. A federal government election on June 12 necessitated postponement of the meeting to June 13 (Fig. 1). Sixteen papers were presented on varied subjects such as: "The Stump and the Prosthetist", "The Dennis-Browne Method of Treatment of Clubfeet", "Fu-

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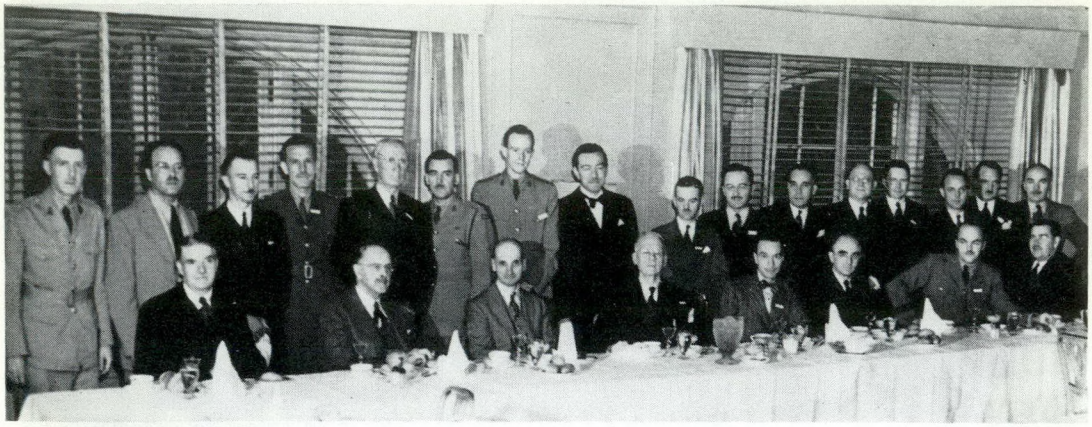

Fig. 1.—The first meeting of the Canadian Orthopaedic Association, Montreal, June 13, 1945. Standing, left to right: Drs. Beath, Rossignol, . . ., Bastedo, . . ., Walker, Bateman, Tremblay, Pouliot, Denoncourt, Goldman, Shapiro, MacLachlan, Townsend, McGibbon and Ewart. Sitting, left to right: Drs. Dale, Turner, Samson, Nutter, Favreau, Armstrong, Coleman and Huckell.

sion of the Adult Hip for Tuberculosis". A symposium on back pain was part of the program. The termination in that year of the war with Germany (May) and Japan (August) made the choice of this date singularly appropriate for already some orthopedic surgeons had returned from overseas and were able to attend this meeting.

At this meeting a draft of the by-laws was submitted, a report of the work of the Provisional Executive Committee was read by Dr. Favreau, and the decision was reached to seek incorporation of the Association through a federal charter as a national bilingual organization with headquarters in Montreal. Letters Patent incorporating the Association were issued by the Secretary of State for Canada in 1948. The requirement for founding membership was set as 10 years in the active practice of orthopedic surgery. It was agreed that from that date officers of the Association were to be elected and business was to be conducted in accordance with the by-laws. In the early years much time was spent in compiling and revising the by-laws. They were finally completed and published in French and English in 1956.

The Association has met annually since 1945 to present a scientific program and conduct appropriate business. The first meetings were held in conjunction with the meetings of The Canadian Medical Association, in the same city and in the same hotel. Later they were held in the same locality as, but immediately before or following the meetings of The Canadian Medical Association. Recently the time of the meeting has remained the same but the location has been different. In 1969 the Canadian Orthopaedic Association held its twenty-fifth annual meeting from June 15 to 19 in Hamilton, Ontario.

The Canadian Orthopaedic Association has been supported by the majority of orthopedic surgeons in Canada and has served as the focus of orthopedic activity and progress in this country. The membership has grown from about 25 in 1945 to 77 in 1948 and to 300 in 1968. The executive committee includes chairmen of some 12 committees and the annual meeting provides a satisfactory opportunity to further the committee work, to have the membership present new work in orthopedic surgery and to record experiences in the diagnosis and management of clinical problems. The quality of the programs equals that of other national orthopedic societies.

Close association has been established with the British Orthopaedic Association, the American Orthopaedic Association, the American Academy of Orthopaedic Surgeons, the International Society of Orthopaedics and Traumatology and the Royal College of Physicians and Surgeons of Canada.

Joint meetings of the Canadian, British

and American Orthopaedic Associations have been held, in 1948 in Quebec City, in 1952 in London, in 1958 in Washington and in 1964 in Vancouver. The next combined meeting of these associations with members of the orthopedic associations of South Africa, New Zealand, Australia and France is to take place in Sydney, Australia, in April 1970. These meetings, with program contributions from surgeons of all these countries, have been a time of excellent fellowship and sharing of experiences. In consequence orthopedic surgery has received benefit and made progress in all these countries, and such gatherings will likely continue at intervals of six years.

At the Quebec meeting in 1948 a program of exchange fellows was initiated. This had its origin in an awareness that in Great Britain, at that time, there were many young orthopedic surgeons who, because of their war service, had never been able to visit Canada or the United States and because of postwar difficulties were not likely to be in a position to do so in the foreseeable future. There was a danger that they would reach senior positions in England with little knowledge of the state of orthopedic surgery in North America. The joint meeting of the American, British, and Canadian Orthopaedic Associations in that year provided an opportunity to bring to Quebec 13 of these young orthopedic surgeons from Great Britain. After attending the meeting and contributing to the program, they travelled across Canada and the United States visiting various centres of orthopedic activity.

This project proved so successful that the British Orthopaedic Association reciprocated in the following year by inviting 15 young orthopedic surgeons (five from Canada and 10 from the United States) to Britain. This also was a great success. They travelled about England and Scotland in a bus (which they called the deep freeze for reasons that will be obvious to Canadians and Americans) and they learned what was being done in orthopedic surgery in Great Britain.

The success of this transatlantic venture was such that discussions were opened among the three associations to establish a continuing program of exchange fellowships. In 1954 the exchange fellowships were renewed for a period of four years with a reduced number of fellows: five from Great Britain and five from North America (four from the United States and one from Canada), in alternate years. This program has been renewed every four years and now includes six from Great Britain and six from North America including two from Canada. To date, 123 young orthopedic surgeons have had an opportunity to take part in this exchange.

Another important accomplishment of the Association was the establishment of the Canadian Orthopaedic Charitable Organization, conceived by Dr. J. L. McDonald, the President in 1955. The purpose was to accumulate funds (from members, from grateful patients and from other sources) which could be utilized for the advancement of orthopedic surgery. The Association approved the idea, which led to the establishment of a trust for receiving and distributing funds for charitable and educational purposes related to orthopedic surgery.

Under the guidance of Dr. R. I. Harris, the original charter of the Canadian Orthopaedic Charitable Organization was surrendered and a new Canadian Orthopaedic Foundation was incorporated as a separate, autonomous, charitable organization under Letters Patent from the Province of Ontario on June 1, 1965. This new foundation undertakes educational programs, sponsors research and maintains liaison with research bodies in orthopedics. It supports the exchange fellowship, the Edouard Samson Prize, the Ian Davidson Memorial Lectureship, the R. I. Harris Memorial Lectureship and its offspring the Canadian Orthopaedic Research Society, which in turn conducts an annual meeting, at which research papers are presented, and sponsors an annual research project.

After 25 years the founders of the Canadian Orthopaedic Association can be satisfied with their undertaking. Orthopedic surgery has become a well-established specialty in Canada with the continual absorption of newly qualified fellows into communities and university centres from coast to coast. Appropriately, the Association is actively engaged in studies on the

education and qualifications of orthopedic surgeons, although official responsibility for these matters rests with the Royal College of Surgeons of Canada. Additional training centres have been approved and examination studies have been undertaken in co-operation with the Royal College. Research co-operation and progress is to be expected from the activities of the Canadian Orthopaedic Research Society. The care of patients and the practice of orthopedic surgery will be furthered by future meetings and programs of the Association. The increase of orthopedic publications in diverse journals will soon warrant their contribution to a Canadian journal of orthopedic surgery. The founders of this Association would likely agree with E. H. Chapin that "The golden age is not in the past, but in the future; not in the origin of human experience, but in its consummate flower; not opening in Eden, but out from Gethsemane."

Acknowledgment is made to the publication "A History of the Canadian Orthopaedic Association" by R. I. Harris, printed by University of Toronto Press for the Canadian Orthopaedic Association in 1967.

PRIMARY INTERNAL FIXATION IN OPEN FRACTURES OF THE LEG

A clinical analysis of 272 patients with either open or closed fractures of lower leg bones led to the conclusions that wound infections and other complications in the healing of fractures are dependent on the character and type of the fracture (which is dependent on the mechanism of injury); that the danger of infection rises with the amount of devitalization, hematoma, and necrotic tissue found at the time of operation; and that oblique and spiral fractures are usually caused by an indirect mechanism and thus the damage to soft tissue is usually not as great. The result is that these rarely become seriously infected, if treated primarily with open fixation. Transverse fractures are usually the result of a direct injury with damage to soft tissues surrounding the fracture. Infections and other complications are infrequent.

Contraindications in the treatment of open fractures by primary internal fixation are the lack of experience of the surgeon; penetrating wounds of irregular extent and large hematomas in the subfascial area, and comminuted fractures. In addition patients in shock and elderly patients are excluded from this treatment.

Only unstable, oblique or spiral fractures with a linear wound, which is not too large, in the patient with good overall condition, skin not too severely damaged, without other pathologic changes such as eczema, without varicosities, and without large abrasions, are amenable to primary internal fixation after open reduction.—Poupa, J.: Zur Frage der primären Osteosynthese offener Unterschenkelbrüche, *Zbl. Chir.*, **94:** 502, 1969.

RADIOLOGIC CHANGES IN THE SHOULDER IN CHRONIC POLYARTHRITIC RHEUMATISM

The shoulder is the most mobile and the most fragile of all the joints in the human body. The muscles and tendons surrounding the shoulder joint and the joint itself show early pathologic changes in degenerative arthritis as well as inflammatory arthritis, particularly in chronic polyarthritic rheumatism. Frequently, changes in the acromioclavicular joint are also associated with these conditions. The authors studied 149 patients—104 females and 45 males. The average age was 57 years and the age ranged from 16 to 77 years.

Arthritic changes of the glenohumeral joint are compatible with changes usually found in other joints in polyarthritic rheumatism. The various changes comprise osteoporosis; subchondral rarefaction of the head, the glenoid and along the insertion of the capsule; narrowing of the joint space; osteophyte formation; and condensation of the articulating surfaces. In most instances, arthritis of the glenohumeral joint is associated with anomalies found in the acromioclavicular joint. Associated with these bony changes affecting the shoulder joint are degenerative processes usually found in the musculotendinous structures within the subacromial tunnel.

In instances of chronic polyarthritic rheumatism, the changes observed in the shoulder include degenerative changes within the rotator cuff in most instances. Frequently, the degenerative process within the rotator cuff causes the rupture of the cuff itself.—Amiel, M. and Graber-Duvernay, B.: Les signes radiologiques de l'atteinte de l'épaule dans la polyarthrite chronique rhumatismale, *J. Radiol. Electr.*, **50:** 7, 1969.

ORIGINAL ARTICLES

PHARYNGEAL DIVERTICULUM: CASE REVIEW AND OPERATIVE TREATMENT*

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PHARYNGEAL diverticulum is not common. MacMillan[1] in 1932 found such diverticula in only 1.8% of patients complaining of dysphagia, and Shallow and Clerf[2] in 1948 reported an incidence of 1 in 1400 hospital admissions. This paper describes 22 patients with pharyngeal diverticula seen at University Hospital, Saskatoon, over a 12-year period from the beginning of 1956 to the end of 1967, an incidence of 1 in 7000 admissions and 1 in 1000 upper gastrointestinal radiographic studies done during these 12 years.

Although this condition is known as Zenker's diverticulum, it was first described by the English surgeon Ludlow in 1769.[3] Not until 1877 did Zenker and von Ziemssen[4] publish their paper, reviewing 22 case reports from the literature and adding five of their own. Since then over 1000 such cases have accumulated in the literature and during the interval therapy has been simplified and made much safer.

In this report we wish to emphasize two points: that a Hurst bougie passed at the time of excision ensures adequate pouch resection and prevents stricture; and that some of these pouches, even of moderate size, can be asymptomatic.

CLINICAL REVIEW

As a preliminary to the study of pharyngeal diverticula, we reviewed all esophageal diverticula recorded at University Hospital over the same 12-year period (Table I). The frequency of mid-esophageal diverticula suggests that some of these patients had previous tuberculous hilar lymphadenopathy. There were six epiphrenic diverticula.

TABLE I.—ESOPHAGEAL DIVERTICULA FOUND IN PATIENTS IN UNIVERSITY HOSPITAL, SASKATOON, 1955–1967

Type	*Number of patients*	*Male/female ratio*
Mid-esophageal	36	21/15
Pharyngeal	22	17/5
Epiphrenic	6	6/0
Multiple	1	0/1
Total	65	

Of the 22 patients with pharyngeal diverticula, 17 were men and five were women. The youngest was 39 years old and the oldest 85 years. The average was 67 years. Such diverticula appear to affect men more than women and their development appears to be associated with the ageing process (Table II).

TABLE II.—PHARYNGEAL DIVERTICULA AT UNIVERSITY HOSPITAL, SASKATOON, 1955–1967

Number of patients	22
Male/female ratio	17/5
Age range (years)	39-85
Average age	67
Symptomatic	16
Asymptomatic	6

Most of these patients had had their symptoms for more than one year (Table III), but 6 of the 22 were entirely without symptoms—a fact that has not heretofore been stressed in the literature. In these asymptomatic patients the defect was discovered on routine barium meal examina-

TABLE III.—DURATION OF SYMPTOMS

Duration	*Number of patients*
Less than 1 year	4
1–5 years	8
Over 5 years	4
Asymptomatic	6
Total	22

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tion done to investigate epigastric distress, or when a barium swallow was administered to assess cardiac size. All but one of these diverticula were primary; one was recurrent, the patient having been operated upon elsewhere.

TABLE IV.—Signs and Symptoms

	Number of patients
Dysphagia	10
Regurgitation	8
Choking	5
Hoarseness	4
Regurgitation by pressure on side of neck	3
Bulge on side of neck	3
Weight loss	3
Nocturnal coughing	2
Gurgling sounds	1

Table IV sets forth the symptoms and signs in these 22 patients. In previous series dysphagia was a prominent symptom, being recorded in 90% to 100% of patients,[5, 6] but in this series dysphagia occurred in less than half the patients. In other series 57% of patients had weight loss, but this loss occurred in only 14% of our patients, probably because they sought medical advice earlier and had mild symptoms. Palmer[7] recorded gurgling noises in the neck as the symptom third in order of frequency in his series; in our group only one patient noted such noises.

TABLE V.—Treatment of Pharyngeal Diverticulum in 22 Patients

Treatment	*Number of patients*
Excision of diverticulum	12
No operation	9
Dilatation	1
Total	22

With respect to treatment only 12 of the 22 patients had excision of the diverticulum (Table V). Six had no symptoms and therefore did not require operation.

Of the other three who were not operated upon one, a 73-year-old man, was considered a poor operative risk. However, this was an error of judgment because he lived four years and, during this time, tolerated a partial gastrectomy for upper gastrointestinal bleeding. He should have been operated upon when first seen because he had progressive dysphagia, which led to increasing weight loss and emaciation. He died of a skull fracture and cerebral damage sustained in a fall, which was probably due to his weakness and emaciation. In general, diverticulectomy is a simple operation, well tolerated by elderly people and, in most instances, should be done if required because the symptoms cannot improve and the patient's condition usually deteriorates. Another patient, a 50-year-old man, had only mild symptoms, but had severe myocardial ischemia. The third patient, aged 80, had complained of mild dysphagia intermittently for three years and several choking attacks over the previous five days. Before admission he had been unable to swallow any food, but on the day he was admitted he regurgitated some undigested food which had probably lodged in the diverticulum. A barium swallow showed a small pharyngeal diverticulum. When esophagoscopy was performed, we could demonstrate no residual obstruction so he was discharged. On follow-up nine years later we found that he had died, but he had had no further trouble from his diverticulum in the intervening years.

An 84-year-old woman, who complained of food sticking in her hypopharynx repeatedly, was treated by dilatation alone. A barium swallow showed a small diverticulum. Esophagoscopy was done and the area dilated. At follow-up she could not be traced.

Technique of Diverticular Resection

Diverticulectomy, the procedure of choice for symptomatic patients, was carried out through an incision along the anterior border of the sternomastoid muscle or through a curved transverse incision along Langer's skin lines. The latter incision is preferable because it gives adequate exposure and a better cosmetic result. The sternomastoid muscle and carotid sheath are retracted laterally and the thyroid gland, trachea and esophagus are drawn medially. The superior belly of omohyoid muscle may have to be divided,

but sternohyoid and sternothyroid muscles can usually be retracted medially by incising the fascia along their posterior borders. Usually, the middle thyroid veins and the inferior thyroid artery have to be divided.

Once the diverticulum has been exposed by rolling the larynx to the side, it is cleared of fascia and areolar tissue until its neck is clearly displayed. At this point a Hurst esophageal mercury-weighted bougie (size 34 to 36) is passed down through the pharynx, and guided by the surgeon into the esophagus. The diverticulum is then excised either by placing a clamp across its neck and cutting across distal to it, or by cutting through the neck with a pair of scissors without a clamp *in situ*, so that the lumen of the esophagus is exposed. The neck of the diverticulum is then closed with either a continuous fine chromic catgut suture or fine interrupted silk sutures. The adjacent muscle and fascial tissue is then approximated over the mucosal closure to obliterate Lannier-Hackerman's space and so reduce the chance of recurrence. In some instances we divide the cricopharyngeus muscle vertically in the midline to eliminate spasm and so prevent recurrence. (Some believe that achalasia of this muscle may be the underlying cause of the diverticulum.) A small Penrose drain is left down to the closure site in the pharynx. We wish to stress the importance of the Hurst bougie; it defines exactly the lumen of the esophagus for the surgeon so that he removes neither too much nor too little of the mucous membrane.

In the patients in this series we had two complications: one recurrence and one leak. The patient with recurrence was operated upon two months later. The leak was temporary and healed completely after 12 days.

One patient with a stricture, an 80-year-old man, had had a diverticulectomy in another hospital. Since the operation he had been unable to swallow properly and, on admission to University Hospital 1½ months later, had a marked stricture in the hypopharynx. Dilatation did not relieve his symptoms, and eventually we did a pharyngoplasty with satisfactory results.

One 85-year-old patient died of myocardial failure three days after operation. He had had previous episodes of congestive failure and had been taking digitalis for several months before the operation.

Follow-Up

We contacted 10 of the 12 patients who had had diverticulectomy and questioned them about their symptoms. The other two had died of unrelated causes. A follow-up barium swallow was performed on these 10 patients. Three had some discomfort on swallowing, but only one had a recurrence (Table VI). Of the six who had no symptoms originally, three were still alive and none had developed any symptoms after the diagnosis had first been made, up to seven years before.

TABLE VI.—Treatment and Results in 12 Patients with Pharyngeal Diverticulum

	Number of patients
Diverticulectomy	12
Complications:	
Recurrence*	1
Fistula (temporary)	1
Death (3 days after operation)	1
Follow-up:	
Free of symptoms	7
Discomfort on swallowing:	3
recurrence*	1
Died of unrelated causes	2

* Same patient.

Discussion

These 22 patients with pharyngeal diverticulum were seen over a 12-year period. They are reported for two reasons: (1) To emphasize the frequency of asymptomatic diverticula, discovered accidentally. That diverticula of considerable size can cause no symptoms has not been noted in the literature previously. (2) To urge others to use a solid esophageal bougie, such as the Hurst mercury-weighted type, when the diverticulum is excised, so that the neck of the pouch can be defined exactly. Beardsley[8] made a similar suggestion in 1961. By using such a bougie the surgeon can steer between the Scylla of recurrence and the Charybdis of stricture formation.

SUMMARY

Twenty-two patients with pharyngeal diverticula were seen over a 12-year period at the University Hospital, Saskatoon. Six of the 22 had no symptoms. The complication rate of excision is low and the cure rate is satisfactory. The operation has been described and the authors recommend that an esophageal bougie be used at the time of operation.

The authors would like to thank Professor H. P. Kent and Dr. C. S. Houston of the Department of Radiology for their assistance in the radiological studies.

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RÉSUMÉ

Les auteurs ont passé en revue les 22 cas de diverticule pharyngé qui ont été traités à l'Hôpital universitaire de Saskatoon au cours d'une période de 12 ans. Ils attirent l'attention sur les trois points suivants: (1) les diverticules du pharynx peuvent être asymptomatiques. Six des malades en question étaient dans ce cas et ne devaient pas être opérés; (2) les malades qui présentent des symptômes devraient être opérés, même s'ils constituent de mauvais risques opératoires; (3) au moment de l'opération, on devrait introduire une bougie rigide dans l'œsophage sous vision directe, de façon à déterminer avec précision la localisation et la dimension du col du diverticule. Ceci permettra d'éviter un rétrécissement excessif au niveau de la fermeture ou, au contraire, de conserver une trop grande portion du col et, partant, d'éliminer le risque de récidive.

COEXISTING CARCINOMA OF THE LUNG AND PULMONARY TUBERCULOSIS

This review of 27 men with coexisting pulmonary tuberculosis and primary carcinoma of the lung confirms other reports which have indicated that one view—that these diseases are mutually antagonistic—was in error. These patients, ranging in age from 42 to 77 years, were seen during a 17½-year period in an institution that treated 6662 patients for pulmonary tuberculosis and 316 for carcinoma of the lung during the same period. Carcinoma and tuberculosis occurred in the same lung in 25 patients. Tuberculosis, which was bilateral in 21 patients and unilateral in six, was far advanced in 10 patients, moderately advanced in 15, minimal in two, and thought to be inactive in 13. Tuberculosis was diagnosed several years before carcinoma developed in 18 patients, and both diseases were found within the same year in nine patients.

Tuberculosis tends to mask a developing carcinoma until the tumour no longer is resectable. At the time of this study, 26 of these patients were dead, and the condition of the one survivor was terminal. Bilateral pulmonary tuberculosis may make the problem particularly difficult and may even lead to thoracotomy on the wrong side. Timely diagnosis of carcinoma of the lung in patients with pulmonary tuberculosis depends upon a high index of suspicion, periodic radiologic follow-up and cytologic tests of sputum, and aggressive work-up and early thoracotomy in patients over 40 years of age who do not respond to antituberculosis chemotherapy. The development of an energic skin reaction to tuberculin in only 1 of these 27 patients indicates that this is an unreliable diagnostic sign. Twenty-four patients gave a history of smoking.—Gerami, S. and Cole, F. H.: Coexisting carcinoma of the lung and pulmonary tuberculosis, *Ann. Thorac. Surg.*, **7:** 317, 1969.

PERFORATION COMPLICATING ADENOCARCINOMA OF COLON AND RECTUM*

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ALTHOUGH perforation with the development of generalized peritonitis is the most dramatic complication of carcinoma of the colon, information concerning it in the English surgical literature is surprisingly scanty.

Unfortunately authors do not always distinguish patients with generalized peritonitis from those with a localized abscess.[1-4] Some reports include cases with fistulas and adherent tumours[5, 6] and also those with perforative diverticulitis.[2, 4]

Because free perforation in malignant disease of the colon is relatively uncommon (0.6% to 4.7% of patients),[6, 7] as is local perforation with abscess formation (0.8% to 4.0%),[5, 8] individual surgeons cannot gain a meaningful personal experience of these conditions. They have difficulty in deciding whether immediate surgical resection has a place in the treatment of these seriously ill patients.

Therefore, the recent experience at Ottawa Civic Hospital was reviewed to determine the important features of the natural history of perforation of the carcinomatous colon, in order to develop a rational plan of management.

MATERIAL AND METHODS

The charts were reviewed of a consecutive series of 1140 patients admitted between 1955 and 1967 to Ottawa Civic Hospital with a diagnosis of adenocarcinoma of the colon and rectum. In all cases complicated by perforation its occurrence was confirmed at operation or autopsy.

Only limited follow-up was available for patients seen before 1959. Therefore, the one-, three- and five-year survival rates for all patients with adenocarcinoma of the colon and rectum (Table I) are based on the follow-up records of the patients registered at the Tumour Registry of the Ottawa Civic Hospital during the years 1959 to 1963 inclusive.

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TABLE I.—SURVIVAL RATES OF PATIENTS WITH ADENOCARCINOMA OF COLON AND RECTUM, OTTAWA CIVIC HOSPITAL

	Alive at 1 year (%)	*Alive at 3 years (%)*	*Alive at 5 years (%)*
With perforation and generalized peritonitis	33	20	12
With local abscess	57	33	25
Tumour Registry patients	63	43	32

RESULTS

Perforation with generalized peritonitis occurred in 28 patients (2.5%) while a local abscess was diagnosed in seven (0.6%).

TABLE II.—SEX DISTRIBUTION OF PATIENTS WITH ADENOCARCINOMA OF COLON AND RECTUM

	Male	*Female*
With perforation and generalized peritonitis	12	16
With perforation and local abscess	1	6
All patients	522	618

The sex and age distributions of patients with perforation were similar to those for the whole series of cases of adenocarcinoma of the colon and rectum (Tables II and III).

TABLE III.—AGE DISTRIBUTION OF PATIENTS WITH ADENOCARCINOMA OF COLON AND RECTUM

	20-39	*40-49*	*50-59*	*60-69*	*70-79*	*80+*	*Total*
With perforation and generalized peritonitis	1	3	7	5	7	5	28
With local abscess	0	0	2	3	0	2	7
Died within 30 days of perforation	0	1	2	2	4	4	13
All patients	43	110	240	323	300	124	1140

The location of the lesions associated with perforation and of all single tumours is set forth in Table IV. That perforation of a rectal tumour was rare is because the complication is anatomically possible only in those close to the rectosigmoid junction (included in the rectal tumours). There were few perforating tumours proximal to the transverse colon. The actual perforation was usually at or immediately adjacent to the tumour. This was true in all patients in whom a local abscess formed after the perforation. In only four of the patients with generalized peritonitis was the perforation found some distance proximal to the tumour (three in the ascending

TABLE IV.—Location of Adenocarcinomas of Colon and Rectum

	Ascending cecum	*Hepatic*	*Transverse*	*Splenic*	*Descending*	*Sigmoid*	*Rectum*	*Total*
With perforation and generalized peritonitis	4	0	6	2	0	9	7	28
With local abscess	0	0	1	0	2	2	2	7
All single tumours	184	38	66	42	69	229	487	1115

colon and cecum and one in the sigmoid colon).

Contrary to the claims of others,[1, 5] preceding symptoms in these patients were not of long duration. Only 8 of the 28 patients with generalized peritonitis and three of the seven with local abscess had symptoms for longer than four months; nine and three patients of these respective groups had symptoms for less than a week. Of all patients reviewed 42% had symptoms for more than four months.

It is apparent, therefore, that as regards age and sex incidence (Tables II and III), duration of symptoms, resectability rate and incidence of lymph-node metastases

TABLE V.—Resectability Rates and Incidence of Lymph-Node Metastases in Patients with Adenocarcinoma of Colon and Rectum

	Resectability rate (%)	*Incidence of lymph-node metastases* (%)
Survivors of generalized peritonitis	75	50
Tumours with local abscess	83	33.3
All single tumours	78	45

(Table V), tumours complicated by perforation are not essentially different from other cancers of the colon. There was a tendency for perforative cancers to be found from the transverse colon to the rectosigmoid junction, no doubt owing to the greater likelihood of obstruction associated with tumours in the distal part of the colon.

Clinical Recognition of Free Perforation

The diagnosis of perforation of a malignant tumour of the colon was rarely made before operation.[1, 9] In cases where the colon was identified as the organ responsible for the clinical picture, diverticulitis was the usual preoperative diagnosis (Table VI).

The mode of clinical presentation significantly influenced the immediate outcome. Of those patients who presented with a "perforated viscus" or "acute generalized peritonitis" only three died. One of these was aged 85 years and died on the thirtieth day after transverse colostomy. Another, an 82-year-old patient, died 12 hours after suffering a cardiac arrest during the induction of anesthesia. The third, a 77-year-old man, had congestive cardiac failure and atrial fibrillation, and died one week after colostomy and drainage.

There were two deaths among the five patients who were thought to have acute appendicitis. One of these, an 81-year-old woman, was treated conservatively for appendicular abscess. The other was 72 years old and was mistakenly treated by appendectomy. In both patients the correct diagnosis was only made at autopsy.

One patient who was being investigated for hepatomegaly suddenly went into shock and died. The diagnosis of perforation was made after an autopsy which also revealed multiple metastases.

The group deserving special mention comprised the eight patients admitted with the diagnosis of "bowel obstruction", of whom six died. It was disturbing to find that in five of these perforation occurred in hospital, either while they were being treated conservatively or while they awaited semi-elective resection. One patient improved sufficiently to undergo transurethral prostatic resection; perforation occurred while he was being prepared for bowel surgery.

Mortality Rates

Mortality was greater in patients with generalized peritonitis (12 of 28 died) than for those with a local abscess (one of seven died).[1] Mortality increased with age (Table

TABLE VI.—Initial Clinical Diagnosis and Associated Mortality in Patients with Perforative Carcinoma of Colon and Generalized Peritonitis*

Diagnosis	*Number of patients*	*Number of deaths*
Perforated viscus	10	3
Acute generalized peritonitis	4	0
Acute appendicitis	5	2
Hepatomegaly and shock	1	1
Bowel obstruction	8	6

*Overall mortality within one month was 43%.

TABLE VII.—Relation of Emergency Treatment of Patients with Perforation and Generalized Peritonitis to Outcome and to the Patient's Condition

Treatment	Number of patients	Immediate deaths	Condition of patient (deaths in brackets)	
			Good	Bad
No operation	3	2	0	3 (3)
Drainage only	2	1	1	1 (1)
Transverse colostomy and drainage	10	4	6 (2)	4 (2)
Appendectomy and drainage	2*	1	2 (1)	0
Ileostomy	1	0	1	0
Ileotransverse colostomy	2	0	2	0
Exteriorization	2	2	0	2 (2)
Right hemicolectomy	3	0	3	0
Sigmoid resection and transverse colostomy	1	0	1	0
Hartmann's procedure	2	1	1	1 (1)

*One patient with a carcinoma of the cecum, perforated appendix and gross generalized peritonitis was treated initially by appendectomy.

III) and, as to be expected, with assignment of an incorrect diagnosis (Table VI).

For patients with generalized peritonitis, mortality appeared to be greatest when conservative therapy was instituted and least when resection was carried out (Table VII). However, this difference was more apparent than real. The treatment was heavily influenced by the patient's general condition.[1] Patients in poor general condition (with shock or toxemia) had a mortality rate of 82% (9 of 11), while of those in good general condition only 18% (3 of 17) died. One of the latter three patients was 85 years old and died on the thirtieth day after operation. In the second, a 72-year-old patient who was mistakenly operated upon for acute appendicitis, the correct diagnosis was not made until autopsy. Thus, survival depended more on the general condition of the patient than on the specific treatment offered.

The only patient with a local abscess who died within 30 days was 81 years old and had an untreated pelvic abscess and carcinoma of the rectum. Four of the six survivors underwent immediate resection and anastomosis, one underwent a three-stage resection, and in one transverse colostomy alone was performed.

Long-Term Survival

Long-term survival rates following perforation with generalized peritonitis were low (Table I), but it must be remembered that 43% of these patients died immediately. The survival rates for patients with local abscess were almost as good as for the Tumour Registry patients.

Discussion

The similar sex and age distributions, the distribution of tumour sites, the similar proportion of patients with long histories, the similar resectability rates and incidence of lymph-node metastases all indicate that tumours of the colon that perforate are little different from other colon adenocarcinomas.[9]

The survival rates of patients suffering perforation and generalized peritonitis are deplorable, a fact noted by other authors.[5, 8] Because of the high immediate mortality rate the long-term survival rates can only be about half of those for other cancers of the colon. Survival rates adjusted for this high immediate mortality are not much lower than those for all colon and rectal adenocarcinomas, so that perforation does not alter subsequent tumour behaviour.

It is important for the surgeon to be aware of the possibility of perforation and to make an early diagnosis when perforation has occurred. A mistaken diagnosis had been made in 8 of the 12 fatal cases among the patients who had generalized peritonitis. In five of these patients perforation occurred in hospital while they were being treated for a bowel obstruction. Presumably this might have been prevented by earlier, more definitive therapy of the obstruction. Probably the patient treated by appendectomy would have survived if the correct diagnosis had been made.[8]

The immediate survival of patients with generalized peritonitis bore such a close relationship to their general condition—rather than to the treatment—that it

would seem unrealistic to subject all patients to primary resection. However, patients treated by this method did so well that primary resection and anastomosis are recommended in all patients whose general condition permits (provided, of course, the lesion is locally resectable).[5, 9] For lesions of the distal colon, the Hartmann procedure might be selected[10] or the anastomosis could be protected with a proximal colostomy.

Although patients with generalized peritonitis admitted in poor general condition have a bad outlook, vigorous resuscitative measures conducted over four to six hours may improve their condition so that some form of bypass and drainage procedure can be carried out. This was the case in two patients in this series who subsequently survived. Considering how rapidly some of the patients in this and other studies[9] deteriorated, it is evident that the resuscitation must be aggressive and rapidly performed.

Patients with local abscess formation can be managed by primary resection if the abscess is small, and by staged resection if the abscess is large.

Conclusions

In a series of 1140 consecutive patients with adenocarcinoma of the colon and rectum, 28 patients (2.5%) had perforation of the colon and generalized peritonitis. Seven patients (0.6%) had perforation with local abscess formation.

In patients with perforative carcinoma of the colon, age, sex and tumour-site distribution were similar to what is found in all patients with carcinoma of the colon and rectum. The proportion of patients who had symptoms for more than four months, the resectability rates and the incidence of lymph-node metastases were also similar. The long-term survival rates were much lower for patients with perforative carcinomas, but this was largely accounted for by the early mortality after perforation.

Early definitive treatment of all bowel obstructions—resection with or without proximal colostomy in all patients with perforation and generalized peritonitis in good general condition, and intense resuscitation of patients in poor condition with subsequent defunctioning and drainage—would appear to yield the most successful results.

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Résumé

Une analyse rétrospective de 1140 cas consécutifs d'épithélioma du côlon ou du rectum a permis de constater que la perforation du côlon est survenue dans 35 cas. Chez sept malades (0.6% du total des cas) s'est produit un abcès local, et chez les 28 autres, une péritonite généralisée. Les malades qui ont présenté cette complication ne différaient en rien de ceux dont la tumeur ne s'est pas perforée. Mise à part la rareté des lésions de l'hémicôlon droit, le développement et l'évolution ultérieure des cancers perforés ont été semblables à ceux qu'on observe dans les tumeurs non perforées du côlon et du rectum. Les principes essentiels sur lesquels doit reposer le traitement de cette complication sont un traitement précoce et radical de tous les cas d'occlusion intestinale, une résection comportant ou non une colostomie proximale chez tous les malades souffrant d'une perforation et d'une péritonite généralisée et dont l'état général est bon, l'application de moyens énergiques de réanimation chez les malades dont l'état général est mauvais avant de procéder à la mise hors circuit et au drainage.

PSEUDOHYPERTROPHIC MUSCULAR DYSTROPHY AND ITS SURGICAL MANAGEMENT: REVIEW OF 30 PATIENTS*

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THE child with pseudohypertrophic muscular dystrophy (Duchenne type) usually comes under medical attention at about 4 years of age because he tires easily, falls frequently, and has difficulty in climbing stairs and getting up from the floor or a chair. As an infant he may have been slow to walk. Although deterioration is insidious, the child's increasing weakness and contractures progressively limit his activities. The relentless progress of the condition continues until compensation is impossible and the child, afraid to walk, subsides into a wheelchair. Limb deformities are then accelerated, collapsing scoliosis develops, and in a few years he is bed-ridden. Finally death by respiratory infection or myocardial failure supervenes in the late teens or early twenties.

Vigorous surgical treatment, properly timed, can modify this sad sequence of events. If, when gait becomes precarious, the contractures are corrected surgically and the limbs braced, the child will be able to continue walking for many months.[1, 2] The withdrawal to wheelchair and bed, with the accompanying deep psychological and social problems, will be delayed.

The purpose of this paper is to report on 105 children with pseudohypertrophic muscular dystrophy seen at The Hospital for Sick Children, Toronto, between 1960 and 1968; to discuss the pathomechanics of their deformities; and, in particular, to evaluate the surgical procedures performed on 30 of these children.

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CLASSIFICATION

The patients in this review were assigned to 10 groups, a modified form of the classification of Archibald and Vignos,[3, 4] and Hanson *et al.*[5] This functional classification (Table I) may not be as accurate as quantitative[6] and qualitative[7] muscle measurements, but we find it reliable and quickly applicable.

TABLE I.—FUNCTIONAL CLASSIFICATION OF PSEUDOHYPERTROPHIC MUSCULAR DYSTROPHY*

Ambulatory patients	*Class*
Climbs stairs: without assistance	1
with help of railing	2
Climbs 8 stairs in over 25 seconds with help of railing	3
Cannot climb stairs: can get up from chair	4
cannot get up from chair	5
Walks with difficulty:	
needs assistance or bracing	6a
uses wheelchair part time	6b
Wheelchair patients	
Moves about independently (bed, toilet)	7
Cannot move about independently:	
can sit up	8
cannot sit up without support	9
Bed patients	
Recumbent, cannot use wheelchair. Needs assistance with all activities	10

*Modified from Archibald and Vignos, and Hanson *et al.*

PATHOGENESIS OF DEFORMITIES

Muscle imbalance, gravity and compensatory postural habits augment each other to produce the deformities. Some authors ascribe the prime role to muscle weakness,[3] others assign it to muscle imbalance, abnormal posture and gravity,[8, 9] and still others believe that gravity and muscle weakness are primarily responsible, claiming that the postural changes are a complex substitution for weak muscles and lead to contractures only secondarily.[10] These views are, in our opinion, not mutually exclusive but complementary.

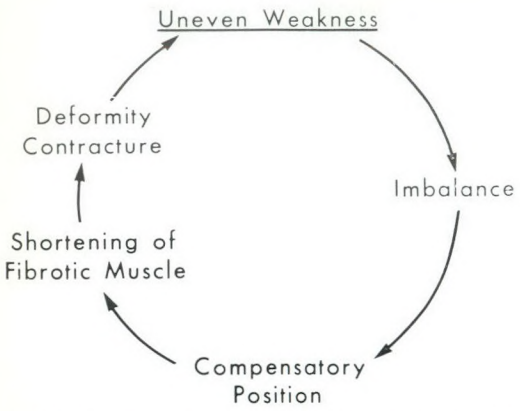

Fig. 1.—The vicious circle of events leading to deformity in muscular dystrophy.

The factors involved operate on one another to create a vicious circle (Fig. 1) where actual muscle shortening and uneven weakness lead to imbalance. To maintain equilibrium the limb compensates proximally or distally by a change of posture creating further contracture and deformity. This produces more weakness, more imbalance and more deformity until compensation is overcome and balance becomes impossible. This is well illustrated in the hip region (Fig. 2). Relatively strong

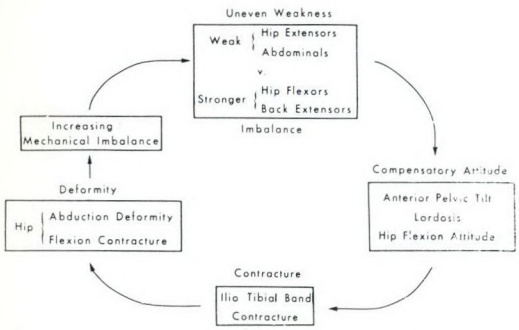

Fig. 2.—The vicious circle as it applies to the development of deformity in the hip region.

hip flexors and spinal muscles are poorly opposed by weak hip extensors and abdominal muscles and favour an anterior pelvic tilt. To maintain erect posture the centre of gravity must be shifted posterior to the weight-bearing axis of the hip joints, and thus lordosis is increased. The hip flexors and the tensor fasciae latae become tighter, producing first an attitude of flexion and abduction of the hip and then a contracture. The flexors gain mechanical advantage over the extensors, muscle imbalance is increased and the circle is complete.

The abduction contracture of the hips helps to widen the stance and maintain equilibrium, just as the equinus helps to prevent jack-knifing of the knees by increasing the tension behind the joints through the origin of the gastrocnemius muscles (Fig. 3). Unfortunately, once any

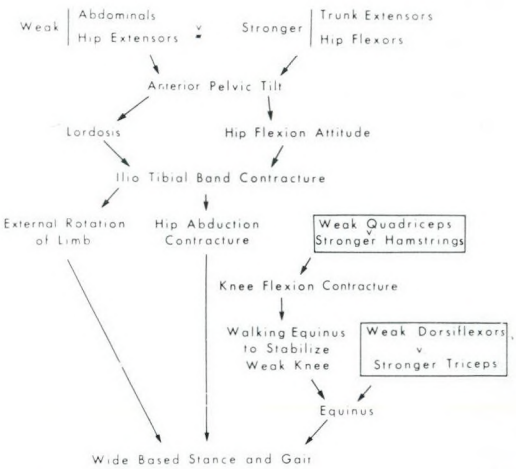

Fig. 3.—Diagram showing the pathomechanics and the sequence of events producing deformities in the lower limbs.

compensatory mechanism is overcome by deformity it becomes a crippling factor. The child stops walking when balance is lost mainly because of the great weakness of the quadriceps and hip extensors, and the marked equinus which makes the base for support unstable.

Aims of Management

Since Duchenne muscular dystrophy is inherited as an X-linked recessive character, the primary aim of management is to prevent its occurrence through the identification of carriers by testing for raised serum creatine phosphokinase levels in female relatives.[11] This provides a basis for genetic counselling. However, by present methods only about 80% of all carriers can be detected.[12] Until effective medical therapy is available, treatment is limited to measures which, although they cannot prolong life, may improve its

quality. These measures include providing special facilities for education, holidays and hobbies; helping parents to provide satisfactory home care, including low calorie diets, and eventually institutional care. Suitable orthotic aids and, where necessary, operations can be used to reduce the severity of deformities where these impair the patient's potential for enjoying the normal experiences of childhood and adolescence.

To achieve this wide goal, all the resources available—the help of neurologist, orthopedic surgeon, physiotherapist, psychologist, social worker, geneticist, public health nurse—must be enlisted. Unfortunately, many of these patients are below normal intelligence (Table II), apathetic and indolent. Special efforts are necessary to motivate the child to co-operate.

TABLE II.—Intelligence Quotients of 65 Patients (all Those who had Formal Assessment). Mean 83

	I.Q. level	*Number of patients*
Bright normal	110-119	5
Average	90-109	15
Dull normal	80-89	14
Borderline	70-79	13
Moderate retardation	50-69	14
Severe retardation	50-↓	4

Active treatment is directed towards delaying deformity by warding off contractures and the progressive disability they impose, thereby interrupting the vicious circle of events leading to loss of ability to walk. Physiotherapy must be initiated early and practised daily throughout the course of the disease.[3, 9, 10, 13] Habitual malposition should be avoided whether the child is awake or asleep. Next, shortened tissues, especially the triceps surae, hamstrings, hip flexors and lumbosacral fascia, require stretching. Success depends on daily active and passive exercises at home directed by the parents, under the supervision of a physiotherapist at least twice a month. Active exercises are good but only temporarily beneficial. Too vigorous exercises do little to strengthen the muscles[14] and, by exhausting the child, may become a burden to him and his family causing frustration and preventing further co-operation when it is more urgently needed.

As weakness increases or contractures become more rigid, bracing should be instituted.[15] Removable bivalved casts or light braces worn at night have little effect and we do not use them. We prefer daytime use of strong long-leg braces, with double upright bars, a knee-lock and limited ankle movement to maintain alignment of weight-bearing joints and provide firm support. These weigh about 1½ lbs. each and are well tolerated.

Surgical Treatment

The purpose of any form of treatment of the dystrophic child is to keep him able to walk. Immobilization of children with muscular dystrophy has deleterious metabolic effects.[16] "The end result of premature use of the wheelchair is a lonely, overweight child whose physical, social, and intellectual development has often been arrested at the level attained when the chair was prescribed."[3] Eventually the weakness becomes so great that the child can remain upright only with the support of braces. In other patients the weakness may be less but the contractures are so deforming that they prevent effective bracing. Since no braces can correct a deformity it must be corrected surgically before the brace is applied. When this is done by simple operative procedures, postoperative immobilization in bed need be only for a day or so and need not interfere with the child's rehabilitation program. Vignos, Spencer and Archibald, by properly timed bracing and operation, increased the length of time a child could walk after his symptoms began from an average of 4.4 years to an average of 8.7 years.[2]

Clinical Material and Procedures

A follow-up study was made of 105 patients seen in the muscular dystrophy clinic of The Hospital for Sick Children during the years 1960 to 1968. Their functional classification at the time of their latest clinic visit is summarized in Table III. Fifty-five per cent are still walking, 34% are wheelchair-bound, 5% are bed-ridden and 6% are dead. Many when first seen were already wheelchair-dependent and a further number progressed to this state

TABLE III.—Status of 105 Patients at Time of Review

	Age (years)																
Class	2.5	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19
1	OO	OO OO	OOO OOO	OO OO	OO													
2			O	OOO	OO OOO			X		X					X		X	
3					O	OO		O		X		X				X		
4								§O										
5								O	§									
6a						§		X	XXXX		§§ XXX	OX X				§		
6b											XX	X						
7							OO		§O XX		§		X OO					O
8											XX §O	X	OO §	XX OO				
9													O	OO X	OO X		O	
10													OO		O	X	O	X
Dead													OO		OOO	X	O	O

O = no special treatment; X = operation ± bracing; § = bracing.

without having braces or operation, because the parents found it easier to care for them in a wheelchair, or because clinic contact was temporarily lost for various reasons.

The patients developed the symptoms of pseudohypertrophic muscular dystrophy between 2.5 and 10 years of age. The mean age at diagnosis was 4.8 years. A few had a history of previous fruitless medical consultations because they were late walking. A positive gastrocnemius biopsy and electromyogram, and elevated serum creatine phosphokinase activity (CPK) were obtained before the diagnosis was accepted. Since 1964 genetic studies using CPK levels have been conducted on the families of all patients. In 44 families the mothers were known to be carriers; of the 65 families containing only a single Duchenne patient, 38 mothers (59%) were shown to be carriers by the finding of elevated CPK activity either in the mother, one of her sisters or one of her daughters. Most of the remaining 27 (41%) patients probably owe their disease to the occurrence of a new mutation in a genetically normal mother, and in these families there is virtually no recurrence risk.[12]

Indications and Techniques

The general indication for operation is the need for correction of a fixed deformity in order to facilitate walking, restore balance, allow bracing, or in a few cases to make the wheelchair patient more comfortable. Ambulatory patients are evaluated every six months; when their gait and balance become precarious, as indicated by greater weakness and more falls, we re-examine them every two months, so that they can be braced or operated on, or both, just before independent ambulation ceases. Muscle strength assessment and serial measurement of joint contracture also help to determine when the time is right for operation. Premature operation may commit the child to wearing braces before he has to; delayed operation may forfeit success, since immobilization for more than a few weeks usually ends independent walking forever.[3]

For the purpose of this study, operative procedures done for these children are grouped under three headings.

(1) *Subcutaneous Tendo Achillis Lengthening* (TAL)

This procedure is done when the unstable support provided by the equinus deformity makes walking precarious. In most of these children equinus was the only deformity, or was the most severe. Where multiple deformities existed operation was sometimes limited to TAL, either because the patient was considered a poor opera-

tive risk for a lengthy procedure, or because his low intelligence might make rehabilitation impossible.

Under tourniquet control we do a subcutaneous tendo Achillis lengthening, not a tenotomy. The tenotome is first introduced at the musculotendinous junction of the soleus muscle on the medial side. The fibres of the tendon are divided half way through their width. A second stab wound is made about 2 cm. above the insertion of the tendon on the calcaneus, and the deeper fibres are divided. The foot is then forcibly dorsiflexed, so that the tendon is elongated. After release of the tourniquet a long-leg cast is applied, with the knees extended and the feet at a right angle. The following day large foam-rubber pads, one inch thick, are applied under the plantar portion of the cast and the physiotherapist gets the child up. The wide bearing surface of the cast allows the child to walk more securely and facilitates his gait-training.

(2) *Achilles Tendon Lengthening, Tibialis Posterior Transfer and Tensor Fasciae Latae Fasciotomy*

This procedure is usually done when bracing to preserve walking is imperative. It is most suitable for the older patient, low in the functional classification and with multiple deformities. The tibialis posterior muscle retains its power for a longer time than other muscles in the region,[1] and is a major factor in producing the equinovarus deformity. Transferring its distal insertion to the dorsolateral part of the foot converts it from a deforming force to a corrective one, that is, from an invertor and plantar flexor to an evertor and dorsiflexor.

First, the fascia lata fasciotomy is performed at three different levels. With the involved hip extended and the opposite hyperflexed, the proximal wound is made near the origin of the tensor fasciae latae from the lateral aspect of the anterosuperior iliac spine; the second wound is made at mid-thigh, and the distal one just proximal to the lateral aspect of the knee joint. The tourniquet is put on, and an Achilles tendon lengthening is performed, as described in (1). Then the tibialis posterior tendon is freed from its distal insertion, transferred through the interosseous membrane and passed through the third cuneiform, where a No. 1 chromic suture anchors it solidly to the sole, over a gauze pad. A long-leg cast is applied after the tourniquet is released and the same postoperative regimen as described in (1) is followed.

(3) *Miscellaneous Procedures*

Most of these are variations of (2), but in one case each, a triple arthrodesis, tibialis posterior division and a hamstring release were also done. The common denominator in this group was an isolated deformity in a patient with a mild form of the disease.

Results

The results are summarized in Tables IV to VI, where the patients are grouped according to the surgical procedure used. The age at the time of operation ranged from 6 to 17 years (mean 9 years), and the follow-up period after operation was from 12 to 100 months (mean 35 months). Operation was done when we believed the children were about to lose their independent walking. In every patient the period of walking was prolonged, for a period of 4 to 100 months with a mean of 25 months.

Two patients developed a moderate hematoma at the wound site in the upper thigh, making rehabilitation more difficult during the first two weeks. Two patients developed superficial pressure sores on one lateral malleolus; these healed quickly and did not delay rehabilitation. All patients had prophylactic preoperative and postoperative respiratory exercises from a physiotherapist, but two developed pneumonia which responded well to penicillin and aerosol therapy. As a result aerosol therapy is now often used in the postoperative period.

All procedures were done on soft tissues, except for a triple arthrodesis in an adolescent (Case 26) whose only deformity was bilateral equinus. Rehabilitation was difficult; at times we doubted whether he would walk again. But now, two years

TABLE IV.—Showing the Findings and Results in Patients who had Percutaneous TAL

Case	Age at diagnosis (years)	Age at operation (years)	Preoperative contracture (mean bilateral)			Postoperative contracture (mean bilateral)			Post-operative follow-up (mos.)	Period of ambulation after operation (mos.)	Functional classification		Long-Leg bracing (LL)	Remarks
			Hip flexion (degrees)	Hip abduction (degrees)	Equinus (degrees)	Hip flexion (degrees)	Hip abduction (degrees)	Equinus (degrees)			Before operation	At review		
1	2½	7	0	0	L 30*	10	10	0	24	24	2	2	nil	Female, L only
2	6	7	0	0	20	10	15	0	78	78	2	2	nil	
3	5	10	0	0	25	0	0	5	72	72	1	2	nil	
4	4	7	15	10	L 25*	15	15	0	30	30	3	6a	LL	L only. LL 8 months after operation
5	5	6	0	10	25	10	15	10	34	34	5	6a	LL	
6	3½	10	0	10	R 40*	30	15	20	30	16	6a	7	LL	LL 3 months before operation
7	5	10	10	20	R 60*	60	45	60	70	6	6a	8	LL	
8	5	12	20	35	40	60	40	5	46	20	6b	8	LL	Wheelchair, 4 months before operation
9	4	9	45	30	R 45*	60	40	R 30* L 60*	44	10	6b	9	LL	
10	3	13	45	30	60	50	40	45	34	0	7	8	LL	Comfort in wheelchair. LL 16 months before operation
11	6	12	45	30	45	60	30	15	40	0	8	9	nil	Comfort in wheelchair

* Indicates degree of unilateral contracture.

TABLE V.—Showing Findings and Results in Patients who had TAL, Tibialis Posterior Transfer and Tensor Fasciae Latae Fasciotomy

Case	Age at diagnosis (years)	Age at operation (years)	Preoperative contracture (mean bilateral)			Postoperative contracture (mean bilateral)			Post-operative follow-up (mos.)	Period of ambulation after operation (mos.)	Functional classification		Long-Leg bracing (LL)	Remarks
			Hip flexion (degrees)	Hip abduction (degrees)	Equinus (degrees)	Hip flexion (degrees)	Hip abduction (degrees)	Equinus (degrees)			Before operation	At review		
12	4	8	10	35	20	25	30	15	23	14	5	7	LL	
13	2½	8	10	40	35	35	30	15	27	20	4	7	LL	LL braces 2 years before operation
14	6	10	10	20	25	10	0	10	25	25	4	6a	LL	
15	4	12	5	30	25	10	20	0	14	14	6b	6b	LL	
16	3	10	5	20	25	10	25	0	14	14	4	3	LL	Walks easily; climbs stairs without braces
17	5	10	10	30	R 25*	0	0	L 15*	24	24	4	6a	LL	R TAL and tibialis posterior transfer; bilateral fascia lata tenotomy
18	4	9	10	25	30	10	15	5	27	27	4	6b	LL	
19	6	9	5	30	25	10	10	10	18	18	3	6a	LL	
20	5	9	20	25	25	5	5	0	12	12	5	6a	LL	
21	6	11	10	40	25	10	10	0	14	14	6a	6a	LL	LL braces 6 months before operation
22	5	10	10	40	20	10	15	0	12	12	5	6a	LL	

* Indicates degree of unilateral contracture.

TABLE VI.—Showing Findings and Results in Patients who Underwent Miscellaneous Surgical Procedures

Case	Age at diagnosis (years)	Age at operation (years)	Preoperative contracture (mean bilateral)			Postoperative contracture (mean bilateral)			Post-operative follow-up (mos.)	Period of ambulation after operation (mos.)	Functional classification		Operative procedure	Long-Leg bracing (LL)	Remarks
			Hip flexion (degrees)	Hip abduction (degrees)	Equinus (degrees)	Hip flexion (degrees)	Hip abduction (degrees)	Equinus (degrees)			Before operation	At review			
23	7	9	0	10	25	10	10	20	66	66	2	3	TAL; tibialis posterior division	nil	I.Q. 50. Minimal surgery for rehabilitation
24	7	11	0	0	40	0	0	0	100	100	2	2	Open TAL; posterior capsulotomy	nil	Female, very mild form
25	5	17	0	0	35	0	0	0	24	24	4	6a	TAL; tibialis posterior transfer	LL on L	Requires cane
26	4	15	0	0	30	0	0	0	22	22	2	3	Bilateral triple arthrodesis	nil	
27	7	11	0	30	25	30	40	5	62	4	5	8	TAL; fascia lata tenotomy	LL	
28	3	14	10	35	0	10	30	0	20	20	5	6a	Fascia lata tenotomy	LL	
29	5	9	15	25	35	30	30	15	30	24	6a	6b	TAL; fascia lata tenotomy	LL	
30	4	14	30	30	30	40	35	40	48	0	7	8	Hamstring release		Female, 60° knee-flexion deformity corrected for bed comfort

later, he walks well without braces. Each of three wheelchair patients had one deformity corrected for the sole purpose of relieving discomfort in bed or in the wheelchair. After operation most patients require long-leg braces.

Discussion

Equinus, present in all but one patient, was the commonest deformity, and the only one in many patients; its severity in the two feet was often unequal. It progressed rapidly in those younger patients with unilateral contracture, in whom early surgical correction was regularly followed by rapid rehabilitation, remarkably slow development of further deformities and often brace-free ambulation. We feel this demonstrates the value of breaking the vicious circle by appropriately timed operation and so delaying the progression of the disabilities due to the disease.

Contrary to our expectation, we found the incidence of recurrent equinus to be almost the same, no matter which operation was used. But in those treated by both TAL and tibialis posterior transfer the equinus recurred later even though the patients tended to be older, to have had more severe deformities and to be already wheelchair-bound when reviewed. Those who had only a TAL usually suffered from a milder form of the disease, often with equinus as the only deformity. We think, therefore, that the tibialis posterior transfer has a real value in delaying recurrent equinus. The longer operative time and more arduous rehabilitation associated with this procedure make it unsuitable for high-risk patients. In these cases we simply cut the tendo Achillis, or the tibialis posterior if there is a varus deformity.

Eighty per cent of all patients showed a hip abduction deformity. Involvement of both the fibres of the tensor fasciae latae and those of the gluteus medius attached to the iliotibial band leads to its tightness.[4] This, among other factors, is responsible for hip abduction and flexion deformity, increased lordosis and anterior pelvic tilt leading to impaired walking ability.[17-19] Section of this band uniformly led to decreased lordosis and hip abduction con-

tracture and, to a minor degree, to decreased hip flexion contracture, and therefore produced a more secure gait.

The recurrence of hip abduction deformity was common, and preceded that of equinus possibly because surgical correction of the hip deformity was less complete. We believe some patients might have been kept walking longer if the recurrence had been corrected again by fasciotomy.

While hip flexion attitude was present in almost every patient, very few walkers actually had a fixed deformity. One-third had no recognizable flexion deformity, and in the others the angle was less than 20°. These findings differ from those of Siegel, Miller and Ray;[20] we may have overlooked this deformity in our earlier patients. However, we feel that flexion deformity is not as incapacitating as abduction deformity and is often partially corrected by tensor fasciae latae fasciotomy. Like Spencer, we have not found it necessary to release the flexors.[20]

Summary

The natural history of pseudohypertrophic muscular dystrophy leads to a vicious circle where weakness, imbalance and deformity act upon one another, increasing the disability until walking is no longer possible.

Operation and bracing, properly timed and part of a rehabilitation program, can break this vicious circle and prolong walking.

Surgical procedures were done on 30 children who by this means were kept walking for an average of 25 additional months, about 10% of their expected life span. These measures have contributed materially to enriching their lives and to reducing the distressing psychological aspects of the disease, both for the patients and for their families.

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Résumé

L'évolution habituelle de la dystrophie musculaire progressive de Duchenne conduit à un cercle vicieux où faiblesse et débalancement musculaires produisent des contractures, puis des déformations qui se potentialisent mutuellement jusqu'à rendre l'équilibre et la marche impossibles.

La chirurgie et l'appareillage des membres inférieurs peuvent interrompre ce cercle vicieux et prolonger la période de marche. Ces mesures doivent faire partie d'un programme de réhabilitation et il est important, à cet effet, que leur indication soit posée au moment opportun — juste avant que l'enfant ne perde sa capacité de marcher — et que les techniques chirurgicales soient peu traumatisantes afin que l'enfant puisse marcher dès le lendemain de l'opération.

Diverses opérations — notamment élongation du tendon d'Achille, transfer du jambier postérieur, fasciotomie du fascia lata — ont permis à 30 enfants de demeurer ambulants pour une période additionnelle moyenne de 25 mois, ce qui représente environ 10% de leur espérance de vie.

Ces mesures modifient le cours de la maladie et retardent quelque peu sa fin inexorable. Elles permettent une existence moins recluse et dépendante, tout en diminuant les effets psychologiques déprimants de la maladie tant pour le patient que pour son entourage.

TOPICAL THERAPY OF BURNS IN CHILDREN

The authors present their continuing experience (from 1964 to 1967) in the care of burns in children, with particular attention to the topical therapy. They report the treatment of 282 children ranging in age from 1 month to 15 years.

During the first three-year period, bulky compression dressings and fine mesh nitrofurazone gauze were used and were changed frequently. In the 1966 to 1967 period the protocol was changed. Therapy in this period differed in that more than one topical agent was used. In this group all patients were treated without dressings except where these were necessary to secure restraints. A topical agent was applied on admission after minimal debridement and cleansing and was applied thereafter two to three times daily. Daily hydrotherapy in a tub on the nursing unit or in a tank was begun as early as 24 hours after injury. Operative debridement was frequently used in large burns to speed the separation of the eschar.

Autografting with sheet split-thickness grafts as well as with mesh split-thickness grafts was performed under general anesthesia. Early hydrotherapy in saline solutions with agitation resulted in a greater percentage of successful split-thickness grafts than a delay in hydrotherapy. There were nine deaths among the first 114 patients and only six deaths in the other 168 patients who had topical therapy.

Mafenide was administered to 89 patients and furazolium chloride was used in 44 patients. When applied topically both agents appeared to suppress bacterial growth. The two agents did not appear to retard eschar separation to the extent that was anticipated. Skin reaction to both drugs developed in a small number of patients.

A total of 282 anesthetics for debridement or grafting was used and there were no deaths from anesthetics and no cardiac arrest. The authors agree that topical antibacterial therapy in children is but one vital factor in the shifting equilibrium between the patient and efforts at recovery and the burn injury and its complications. Colloid in the form of plasma and blood would appear to have a place in the resuscitation of pediatric patients with thermal injury. Various combinations of replacement therapy were used. Ringer's lactate solution and 0.9% sodium chloride were used alone for resuscitation in 28 children; whole blood was used in five; plasma or albumin with electrolyte was used in 32; and a combination of blood, plasma, and electrolyte was used in nine patients.

Penicillin was used routinely in all but minor burns during the first five days to prevent streptococcal infection. It was also used routinely in all patients having split-thickness grafting.

Nine children all under the age of 4 years and with significant burns had major gastrointestinal hemorrhage. Five patients with major gastrointestinal hemorrhage died and in one it led directly to death. The authors agree that surgical intervention in Curling's ulcer should be handled in a manner similar to that used on any other patient with severe upper gastrointestinal hemorrhage. Cardiac failure developed in two children and neurologic complications developed in nine.—Smith, E. I. and DeWeese, M. S.: The topical therapy of burns in children, *Arch. Surg. (Chicago)*, **98:** 462, 1969.

FOCAL NODULAR HYPERPLASIA OF THE LIVER*

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LOCALIZED nodular lesions of the liver resembling tumours have been observed as incidental findings at autopsy since 1884. In that year the condition was first reported by Simmonds,[1] who described it as showing only regenerative hyperplasia. Since then similar lesions have been reported under various names such as adenoma,[2] hamartoma,[3, 4] benign hepatoma,[5] solitary hyperplastic nodule[6] and focal cirrhosis of the liver.[7] Edmondson[8] has recently introduced yet another term, "focal nodular hyperplasia", indicating that the histological features are different from the basic morphology of cirrhosis or neoplasms. Although this entity is uncommon, the surgeon is occasionally confronted with a localized nodular lesion in the liver during laparotomy performed for other reasons. Since the lesions are frequently subcapsular, hard and grey-white, they may be mistaken for metastatic carcinoma. Alternatively, especially in children, the mass may be large enough to suggest a primary hepatic tumour. The recognition of these hyperplastic nodules by the surgeon and awareness of their existence by the pathologist are essential to allow a prompt diagnosis to be made at frozen section and the correct surgical procedure to be instituted. We recently encountered three cases of focal nodular hyperplasia, one as an incidental finding at autopsy and two that presented diagnostic problems at laparotomy.

CASE REPORTS

Case 1.—A 40-year-old woman was admitted to hospital with a 12-month history of episodes of upper abdominal pain associated with eating fatty meals. Radiological examination revealed a solitary radiolucent calculus in the gallbladder. A cholecystectomy was performed and at operation the surgeon noted

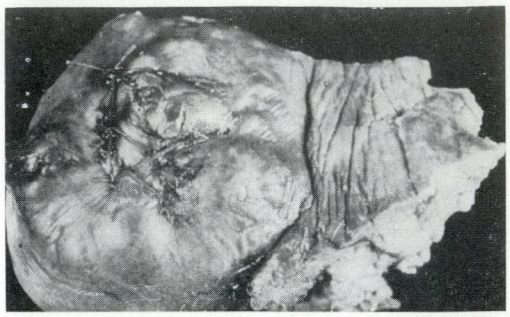

Fig. 1.—External appearance of the resected liver nodule from Case 1. Note the knobby appearance. Sutures indicate the site of the biopsy.

that at the anterior border of the left lobe of the liver was an irregular nodular mass, yellow-tan in colour, measuring approximately 6 x 6.5 cm. (Figs. 1 and 2). Parts of this mass were firm in consistence. A biopsy was performed which showed mild inflammatory changes, and a diagnosis of focal nodular hyperplasia was made. A left hepatic lobectomy was performed. The patient's postoperative course was uneventful. She has been well and free of symptoms for nine months.

Fig. 2.—Cut surface of the liver nodule in the surgically resected specimen from Case 1. Note the subcapsular nature, stellate appearance and distinct demarcation from adjoining parenchyma.

Case 2.—A 34-year-old woman was admitted with a three-month history of right upper quadrant pain which occasionally radiated to the shoulders. This was invariably associated with nausea and vomiting and was exacerbated by fatty meals. Although radiological examination of the biliary tree was negative, a cholecystectomy was performed in view of the clinical history. At laparotomy a solitary sub-

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capsular, firm and umbilicated nodule, 2.5 cm. in diameter, was noted in the left lobe of the liver. This was biopsied and from a frozen section focal nodular hyperplasia was diagnosed. The lesion was then excised *in toto*. The patient's postoperative course was uncomplicated and she has now been well and asymptomatic for five months.

Case 3.—A 30-year-old woman was admitted with signs and symptoms of meningitis from which she eventually succumbed. There had been no evidence of a previous gastrointestinal disorder. An incidental finding at autopsy was a subcapsular grey-white firm mass in the right lobe of the liver measuring approximately 4 cm. in diameter. It was clearly demarcated from adjoining hepatic parenchyma (Fig. 3).

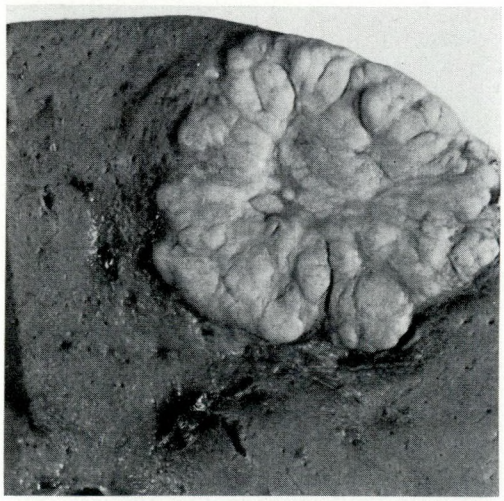

Fig. 3.—Higher magnification of the nodule from Case 3. Several small nodular components of the large mass are evident as is the stellate appearance.

In none of these patients was there a history of exposure to hepato-toxic drugs; the third patient whose case is described received broad-spectrum antibiotics.

Observations

The gross and histological features of the hepatic tumour were qualitatively similar in all three cases and are summarized below.

Gross

In Cases 1 and 2 the left lobe was involved whereas the incidental lesion found at autopsy in Case 3 affected the right lobe of the liver. In the first patient described the tumour presented as a projecting knobby mass on the external surface (Fig. 1). The nodules were subcapsular, solitary, firm and sharply circumscribed from adjoining hepatic parenchyma (Fig. 2). Cut surfaces were a yellow-grey colour with broad fibrous trabecular strands extending from the centre to the periphery resulting in a stellate appearance with a somewhat lobular arrangement (Fig. 3).

Microscopic

The nodules were well circumscribed but did not have a true capsule and were bounded mostly by compressed hepatic parenchyma (Fig. 4). In focal areas this "pseudocapsule" was limited by portal tracts and occasionally by fibrous tissue. These large masses were composed of multiple regenerative nodules some of which were well formed while others were still in the stage of development (Fig. 5). In the portal and central areas fibrosis was marked with tongue-like projections joining adjacent fibrotic foci. These fibrous bands were infiltrated by moderate numbers of lymphocytes (Fig. 6). There was a moderate degree of bile-duct proliferation. There was mild focal fatty metamorphosis, rarely absence of reticular framework around

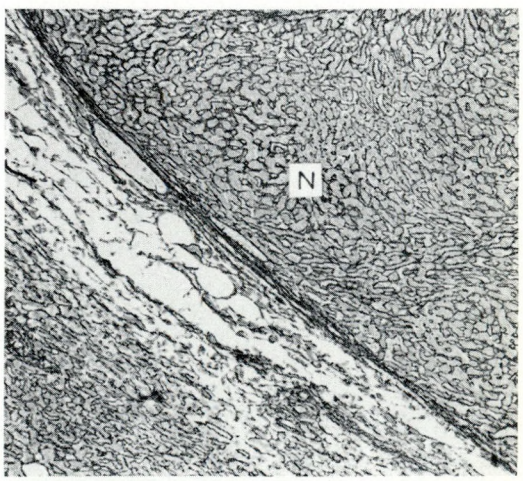

Fig. 4.—Clear demarcation between the nodule (N) and the adjoining hepatic parenchyma with the intervening "pseudocapsule" (Gomori's reticulum stain, original magnification × 50).

Fig. 5.—Regenerative nodules showing fatty metamorphosis. Note the increased fibrosis in the portal areas with fibrous projections encircling the liver nodules (hematoxylin-phloxine-saffron stains, original magnification × 60).

hepatic cells and irregular distribution of glycogen. Mitosis, giant cells and pleomorphism were not in evidence. The adjoining hepatic parenchyma was essentially normal.

Discussion

Focal nodular hyperplasia usually presents as a solitary subcapsular mass, commonly in the right, but occasionally in the left hepatic lobe, as exemplified by two of the cases reported here. Infrequently these masses are seen deep in the parenchyma. A fully developed lesion, illustrated by our cases, is composed of a central stellate area of fibrosis from which thin bands of fibrous connective tissue radiate. A characteristic feature is that these lesions are more conspicuous on gross examination than on microscopic study. They are partially or completely encircled by a pseudocapsule composed partly of the stroma of the adjacent liver. The large masses are composed of irregular regenerative nodules separated by bands of fibrous tissue containing variable numbers of inflammatory cells and showing ductular proliferation. On occasion the central core of the mass is made up of fibrous connective tissue in which proliferating bile ducts and round cell infiltration are conspicuous features.

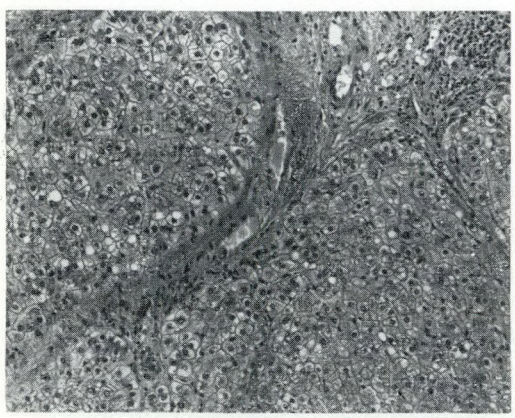

Fig. 6.—Regenerative nodules separated by bands of fibrous tissue infiltrated by moderate numbers of lymphocytes (hematoxylin-phloxine-saffron stains, original magnification × 125).

The etiology, pathogenesis and nature of these lesions are unclear. Arguments in favour of a regenerative phenomenon, a hamartoma or a neoplasm can all be advanced. It has been suggested that until more is known about the disease it be designated as "focal nodular hyperplasia".[8] However, the weight of evidence seems to favour the concept of a regenerative or localized "cirrhotic" process as being responsible for these lesions. Simmonds[1] as early as 1884 noted that nodule formation was a peculiar response of the liver to injury. Furthermore, he discussed the difficulties of distinguishing hyperplastic nodules from adenomas, and adenomas from small encapsulated carcinomas of the liver cell type. In 1950 Kay and Talbert[2] reported two cases of benign primary lesions of the liver which they labelled "mixed type" adenomas or hamartomas. Their first case was that of a 7-month-old girl who had a large solitary mass measuring 9.5 x 7.5 x 6.0 cm. in the caudate lobe of the liver. The second was that of a 13-year-old boy who presented with a large tumour replacing the left lobe and half of the right lobe of the liver. In neither case could the tumour be excised because of its large size. Tissue obtained at biopsy in both cases showed histological features similar to those seen in our three cases and a 7½-year follow-up of the second case disclosed no evidence of recurrence.[2, 8] In their discussion the authors contended that although the "tumour" resembled portal cirrhosis it was probably congenital in origin.

Benz and Baggenstoss[7] reviewed 34

necropsy cases of hepatic lesions which had been labelled hamartomas, adenomas, benign hepatomas and solitary hyperplastic nodules, identified over a period of 30 years (1922 to 1951) at the Mayo Clinic, Rochester. They studied the histogenesis of this lesion from its early formation to its late stages and rejected a neoplastic or hamartomatous concept. Their conclusion was that these nodular lesions were identical to focal areas of cirrhosis, not only in their gross appearance but also in their histological features. The latter included infiltration with fat, alcoholic hyalin, proliferation of bile ducts, fibrosis and sometimes features of a recovery phase of acute hepatitis. They also considered, but could not support with evidence, the possibility that this "focal cirrhosis" may develop in an area with an aberrant blood supply or bile-duct malformation.

Edmondson[8] maintains that neither vascular changes nor necrosis are evident in early lesions. Moreover, bile ducts are usually located at the periphery of pseudolobules in the various types of cirrhosis whereas in many of the component nodules of focal nodular hyperplasia cord cells are arranged in a spherical manner around bile ducts, vessels and connective tissue. For these reasons he considers that it is difficult to accept the concept that these lesions are the result of regenerative phenomena alone.

Although the exact histogenesis remains uncertain and under dispute a distinction must be made between a true adenoma of the liver and focal nodular hyperplasia. Grossly, a characteristic central stellate area is not seen in adenomas. Microscopically, the cells composing an adenoma are often somewhat atypical and there is no trace of bile ducts or portal areas. In focal nodular hyperplasia the cellular morphology is practically no different from that of normal liver and in addition the cells are arranged in relation to bile ducts and accompanying connective tissue.

Focal nodular hyperplasia is more common in women and many of the "tumours" have been seen in children.[9] These "tumours" are usually incidental findings at laparotomy or autopsy but may present as an abdominal mass, particularly in children.[2, 9] This invariably asymptomatic nature is in marked contrast to partial nodular transformation of the liver, an entity recently described by Sherlock *et al.*,[10] which is associated with portal hypertension.

Single nodular lesions of the liver are rare but occasionally confront the surgeon at laparotomy. The lesion may closely resemble secondary tumour, in which case biopsy and examination of a frozen section are helpful in identification and in determining the extent of the surgical excision that will be required. For a definitive diagnosis and to distinguish the tumour from an adenoma and well-differentiated carcinoma it is generally recommended that, if possible, the lesion be resected *in toto*. Since the pseudocapsule may not encircle the entire lesion and residual tissue may be left behind, simple "shelling out" of the mass is not recommended. In the case of large unresectable tumours the surgeon must be content with biopsy; as yet there is no evidence to suggest that such masses become malignant or grow sufficiently to endanger life. However, there are only two patients on record who have remained well after periods of three and 7½ years respectively after the diagnosis was confirmed, so that the latter statement must be accepted with considerable reserve. It is therefore important that cases of focal nodular hyperplasia be reported in order that the growth potential of these lesions can be correctly evaluated.

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RÉSUMÉ

Les auteurs présentent trois cas de masses nodulaires solitaires dans le foie et qu'ils ont identifiées comme étant une hyperplasie nodulaire en foyer. Deux de ces cas ont été découverts au moment d'une laparotomie pratiquée pour une pathologie différente et leur diagnostic a été difficile.

Ces lésions sont rares, mais le chirurgien devrait considérer cette possibilité, étant donné qu'elles peuvent simuler, lors d'une laparotomie, des lésions primaires ou des métastases d'un cancer du foie.

NON-PARASITIC CYSTS OF LIVER

The surgical treatment of three patients with benign cysts of the liver is presented. In all three the cyst wall was partially excised; the results were satisfactory.

The first patient, a 72-year-old woman with the diagnosis of subsiding acute cholecystitis, was operated on in 1949. She had a large cyst arising from the inferior surface of the right lobe of the liver, with several smaller cysts on the dome of the right lobe. The large cyst was aspirated; 600 ml. of clear water-like fluid was removed. The dome of the cyst was excised, leaving its base attached to the liver. The smaller cysts were also aspirated. The patient had no further abdominal symptoms until she developed carcinoma of the colon; she died in 1967, 18 years after the treatment of the liver cysts.

The second patient was a 57-year-old woman who complained of right upper quadrant pain. At laparotomy she had a large cystic mass replacing a major portion of the right lobe of the liver. The cyst was aspirated and 1450 ml. of a greenish fluid was obtained. She was treated in the same way as the first patient and has remained well for 15 years since operation.

The last patient, a 52-year-old man, had a mass in the right upper quadrant and epigastrium. He received the same treatment as the first two patients. The cyst contained 2000 ml. of a clear water-like fluid. The patient is well one year after his operation.

All these patients did well and none had evidence of intraperitoneal bile leakage from the portion of the cyst left within the liver. The aspiration of the cyst aided in the determination of the presence or absence of bile before partial excision of the cyst or internal drainage was elected.—Belcher, H. V. and Hull, H. C.: Nonparasitic cysts of the liver: report of three cases, *Surgery*, **65:** 427, 1969.

HEPATIC RESECTION IN CHILDHOOD

Since 1870, when the first partial hepatic resection was performed, various centres have shown remarkable advances in performing hepatic resections, but experience with infants and children has been limited. In the past 15 years at one hospital centre 22 children underwent partial hepatic resection. Nine had primary malignant neoplasms, four had hepatic lymphangiomas, three had metastatic Wilms' tumours, two had simple cysts, two had hamartomas, one had an adenoma, and one patient required lobectomy for trauma. The age range was from 1 to 8 years. Three operative deaths due to massive intraoperative hemorrhage occurred, one operative air embolus occurred, and two died from metastasis.

Preoperative radioisotope liver scans, hepatic and superior mesenteric arteriograms are helpful in diagnosis and in determining the operative approach. Excessive intraoperative bleeding is a primary concern; immediate replacement with warm fresh buffered blood infused through a cannula in a superior vena cava is essential. Postoperative care includes fluid and colloid replacement with blood, albumin, and dextrose. Irradiation and chemotherapy should be withheld until the period of most active regeneration is over.—Taylor, P. H. *et al.*: Experience with hepatic resection in childhood, *Amer. J. Surg.*, **117:** 435, 1969.

FEMORAL HERNIA FOLLOWING INGUINAL HERNIORRHAPHY*

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THE development of femoral hernia as a complication of inguinal herniorrhaphy has been reported by several authors.[1-9] Clinical reviews of femoral hernia by some of these authors were tabulated by Jones,[8] and to the data that he collected I have added my own (Table I). These reviews all indicate that femoral hernias that have followed a previous inguinal repair form a surprisingly high percentage of all femoral hernias encountered. One in every four patients who develop a femoral hernia has previously undergone an inguinal herniorrhaphy.

TABLE I.—FEMORAL HERNIAS FOLLOWING INGUINAL HERNIORRHAPHY AS A PERCENTAGE OF THE TOTAL NUMBER OF FEMORAL HERNIAS ENCOUNTERED

Author	*Year published*	*Number of femoral hernia operations*	*Number of operations for femoral hernias that followed ipsilateral inguinal herniorrhaphy*	*Percentage*
McClure and Fallis[1]	1939	90	20	22
Fratkin[2]	1948	20	9	45
Ludington[3]	1958	62	26	42
Burton and Bauer[4]	1958	165	46	28
Jones[8]	1966	20	3	15
Glassow[7]	1966	1143	255	23
Total		1500	359	24

In these reviews, however, no figures are available for the incidence of femoral hernia following inguinal herniorrhaphy considered as a percentage of the total number of *inguinal* repairs performed, probably because of its comparative rarity in any single series. Likewise no information is supplied regarding the type of this original inguinal hernia. The purpose of this paper is to provide answers to these questions as well as to discuss the etiology of the condition.

A large series of inguinal herniorrhaphies performed under relatively standard conditions in a single hospital is particularly suitable for this purpose; the present report is based upon experience at the Shouldice Hospital, Toronto, during the 22-year period from January 1, 1945 to January 1, 1967. During this time approximately 45,000 primary inguinal hernia repairs were performed and approximately 5000 recurrent inguinal hernia repairs in patients whose initial repair had been done elsewhere. There were 111 instances of femoral hernia developing after inguinal herniorrhaphy, an incidence of 0.22%. Of these, 109 were in men and two were in women. In 97 of the 111, the preceding repair was performed for primary inguinal hernia and in 14 it was performed for recurrent inguinal hernia. In 75 of the 111 a repair of the femoral hernia has been performed in this hospital while in the remaining 36 no such operation has been done, in most instances because the femoral hernia was small and asymptomatic and the patient was elderly.

Only purely inguinal hernia repairs with this complication are reported. If the original hernia repair was performed for combined ipsilateral inguinal and femoral hernias it has not been included in this series.

During the period reviewed the number of femoral hernia repairs has increased to 1223, because a further 80 repairs have been performed since the series reported in Table I was collated. As well as the 75 operations mentioned above, 862 operations were performed for primary femoral hernia and 286 were performed for femoral hernia

*From the Shouldice Hospital, Toronto, Ont. Based on a paper presented at the Annual Meeting of the Royal College of Physicians and Surgeons of Canada, Vancouver, B.C., January 1969.

TABLE II.—Type of Inguinal Hernia that was Followed by an Ipsilateral Femoral Hernia

Inguinal hernia type	*Number of patients undergoing operation*	*Number of patients developing femoral hernia subsequently*	*Percentage*	*Femoral hernia subsequently repaired*	*Femoral hernia not repaired*
Primary indirect	28,000	46	0.16	33	13
Primary direct	14,000	46	0.33	30	16
Primary indirect and direct	3,000	5	0.17	2	3
Recurrent indirect	2,200	1	0.05	1	0
Recurrent direct	2,300	11	0.48	7	4
Recurrent indirect and direct	500	2	0.40	2	0
Total	50,000	111	0.22	75	36

that had followed an initial inguinal or femoral hernia repair performed elsewhere. In this last group, unfortunately, the operation records were not available, making it impossible to determine the exact distribution of the original hernia types. In this respect the figure "255" in Table I is composite in nature. In 75 cases the femoral hernia repair was performed following a previous ipsilateral inguinal herniorrhaphy in this hospital, while in the remaining 180 cases the surgeon considered it probable, in the absence of records of the previous operation, that the original repair had been for inguinal hernia.

Femoral Hernia Following Primary Inguinal Herniorrhaphy

In this group there were 97 patients (96 men and only one woman), an incidence of 0.19%.

From Table II it can be seen that approximately the same number of patients developed the condition following the repair of an indirect as of a direct inguinal hernia. However, in the 45,000 primary inguinal herniorrhaphies performed in this period there were twice as many indirect as direct inguinal hernia repairs. Hence, femoral hernia develops twice as often following repair of a primary direct as of a primary indirect inguinal hernia. This suggests a strong correlation between the condition and a weak posterior wall of the inguinal canal.

Further support is given to this hypothesis by consideration of the rarity of this complication in women. In an earlier paper[10] the author reviewed the rarity of direct inguinal hernia in women and its relevant corollary, the presence of a strong posterior wall of the inguinal canal. In this series, although they made up about one-twentieth of the patients operated upon for primary inguinal hernia, only one woman, as opposed to 96 men, subsequently developed an ipsilateral femoral hernia, an incidence of 0.05%.

Table III shows that the condition developed in almost twice as many patients after a bilateral rather than a unilateral primary inguinal hernia had been repaired. Numerically, primary unilateral inguinal hernias outnumbered primary bilateral inguinal hernias in the original series of 45,000, the ratio being approximately four to one so that femoral hernia occurred eight times more frequently in a patient who had had bilateral primary inguinal hernia repair.

These statistical observations suggest that tension in the original repair is a factor of considerable importance in the production of femoral hernia.

There was no recorded case of bilateral femoral hernia.

Sixty-five of these 97 patients have undergone repair of the femoral hernia.

Femoral Hernia Following Recurrent Inguinal Herniorrhaphy

There were 14 patients in this group, an incidence of 0.28%. Of these patients 13 were male and one was female. In 10 the femoral hernia has been repaired.

Table II shows that the condition developed much more frequently following a repair of recurrent direct inguinal hernia than it did following a repair of a recurrent indirect inguinal hernia, although in this series of 5000 recurrent inguinal hernia repairs indirect and direct inguinal recur-

TABLE III.—Distribution of Unilateral and Bilateral Inguinal Hernia Repairs Followed Later by Femoral Hernia

Inguinal hernia type	*Number of patients undergoing operation*	*Number of patients developing femoral hernia subsequently*	*Percentage*	*Femoral hernia subsequently repaired*	*Femoral hernia not repaired*
Unilateral:					
Primary	36,000	34	0.09	22	12
Recurrent	4,350	7	0.16	6	1
Bilateral:					
Primary	9,000	63	0.70	43	20
Recurrent	650	7	1.08	4	3
Total	50,000	111	0.22	75	36

rences were present in approximately equal numbers.

Table III indicates that femoral hernia was as frequent following a repair of a unilateral as of a bilateral recurrent inguinal hernia, yet the former type of repair was performed approximately seven times as often as the latter.

From the group of 80 women admitted with recurrent inguinal hernia, itself a rare condition in women, only one developed femoral hernia. The original hernia in her case was direct inguinal in type, as it was in two-thirds of the members of this group.

These facts support the conclusions of the previous section.

Etiology

In the great majority of cases the femoral hernia appeared within three years of the original repair. Both age and physique seemed to be important factors in its development. Only 10% of patients were less than 40 and 75% were more than 50 years old. The majority weighed less than 150 lbs.

A femoral hernia occurring after inguinal herniorrhaphy may arise in three ways: (1) because it was missed at the original repair, (2) because it is a new hernia, or (3) because it was created by the original repair; (2) and (3) may be interrelated. It is considered unlikely that many in this series represent missed femoral hernias. It is the practice of surgeons in this hospital routinely to examine the femoral region at operation and by so doing more than 100 clinically unsuspected femoral hernias have been discovered. In the earlier sections of this article it has been shown that there is a relationship between femoral hernia following inguinal herniorrhaphy and direct inguinal hernia on the one hand and bilateral inguinal repair on the other. Probably the technique used at the original inguinal herniorrhaphy was the main factor responsible. Undue or unnecessary tension in an inguinal repair may weaken or open up the femoral region and so predispose to femoral hernia by pulling the transversalis fascia and the inguinal ligament upwards. Anson and McVay,[11, 12] McVay and Savage,[13] Easton,[1] Fratkin,[2] Ludington[3] and Jones[8] all make similar comment.

Although this condition is now the most frequent type of recurrent hernia following inguinal herniorrhaphy performed at the Shouldice Hospital, because of its low incidence little may be expected from prophylactic measures. There may occasionally be a place for the use of a relaxing incision[14] although this has not been practised here. However, the case in which particular attention to technique is necessary is that of bilateral direct inguinal hernia in the older man of poor physique, where the element of tension in the repair is greater than usual. In such a situation some consideration should be given to staging the two repairs several months apart.

Treatment

In all 75 patients undergoing a repair of a femoral hernia that developed after inguinal herniorrhaphy, the operation was performed from below the inguinal ligament. In only a few of these patients the inguinal region also was opened and ex-

plored. In 4 of the 75 yet another recurrence has developed, a recurrence rate of 5%. In three the subsequent recurrence was femoral and in one it was inguinal. In two of these four another repair has been performed.

The technique for repair of inguinal and femoral hernias used in this hospital has been described elsewhere.[5-7] Regional infiltration anesthesia is routinely used preceded by adequate preoperative sedation. Bilateral repairs are routinely staged 48 hours apart. The comprehensive follow-up system lasts not less than 10 years.

Summary

In a series of approximately 50,000 inguinal herniorrhaphies performed in a 22-year period, 111 cases of ipsilateral femoral hernia subsequently occurred, an incidence of 0.22%; 109 occurred in men and two in women.

The condition followed repair of indirect inguinal hernia in 47 instances, repair of direct inguinal hernia in 57 instances, and repair of an ipsilateral combination of the two types in seven instances.

Etiological factors are discussed, stressing in particular the importance of physique, age, the strength of the posterior wall of the inguinal canal, tension in the repair and the rarity of the condition in women.

The condition occurs most frequently following bilateral direct inguinal hernia repair in the elderly.

A subinguinal operation was performed in all 75 cases undergoing repair of the femoral hernia, with a further recurrence rate of 5% in this group.

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Résumé

La hernie crurale est une complication rare de la herniorraphie inguinale. C'est ainsi que, sur les quelque 50,000 herniorraphies inguinales pratiquées au Shouldice Hospital de Toronto durant une période de 22 ans, on n'en a enregistré que 111 cas, ce qui représente une proportion de 0.22%. Deux de ces malades étaient des femmes et 109 des hommes. Cette complication est survenue deux fois plus souvent après réparation d'une hernie inguinale directe qu'après herniorraphie pour hernie inguinale indirecte. Elle est plus probable après réparation d'une hernie inguinale directe bilatérale chez le vieillard.

Cette complication est probablement la conséquence de la première opération si on considère que, dans la majorité des cas, elle survient trois ans après cette opération. Parmi les facteurs étiologiques figurent l'âge, le sexe, le physique, la faiblesse de la paroi postérieure du canal inguinal et la tension exercée durant la herniorraphie.

Chez les 75 patients qui ont été opérés pour cette complication par une intervention subinguinale, on a noté une récidive dans 5% des cas.

A COURSE IN SURGICAL TECHNIQUE FOR MEDICAL STUDENTS*

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FOR the past eight years an elective 12-week course in surgical technique for undergraduate medical students has been conducted during the summer vacation break at the University of Alberta (Fig. 1). Some 50 students from the first, second and third years of medicine have taken this course which consists of weekly sessions held at the Surgical-Medical Research Institute. All operations are performed on dogs.

Fig. 1.—The "Class" of 1966. Complete gowning and gloving is used for all procedures.

The purposes of the course are threefold: (1) to acquaint medical students with standard surgical techniques; (2) by so doing to help the student to carry out his other summer research program more effectively (most of the students are engaged elsewhere in projects requiring some knowledge of surgical technique); and (3) to evaluate the methods by which surgical technique is learned.

PERSONNEL

The Director of the course is assisted by three or four members of the Department of Surgery, the teaching fellows in surgery and the senior residents in general surgery and the subspecialties of thoracic and plastic surgery, urology and neurosurgery. One laboratory technician is assigned to two operating tables.

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THE COURSE

All procedures are preceded by a 5- to 10-minute orientation talk, followed by a demonstration operation performed by the instructors. The students, working in pairs, then carry out the procedures themselves under supervision. The entire laboratory experience is completed in four hours and most of the operations are carried out as acute experiments. The students are taught the proper method of recording their operative experience and these records are handed in, corrected and returned.

At three or four stages during the course, cinematic records are made of each operating surgeon. These films are projected repeatedly for the benefit of the instructors and the student.

Once weekly, on a separate occasion and using the actual operation as a topic, a short course in medical communication is conducted. During the course each student must give two 10-minute talks using all the recognized techniques of a good scientific presentation.

An operating manual has been prepared for the student's use and to this he adds his corrected operative reports and his prepared talks.

THE 12 OPERATIONS

(1) General principles including sutures, knots, incisions and instruments.

(2) The sterile technique. This is taught at the University of Alberta Hospital where the student scrubs with the instructors.

(3) Surgery of the thyroid and parathyroid.

(4) Vascular surgical technique.

(5) Biliary tract surgery.

(6) Surgery of the stomach (1).

(7) Surgery of the stomach (2).

(8) Gastrointestinal surgery.

(9) Renal transplantation.

(10) Pulmonary surgery.

(11) Tendon repair and skin grafting.

(12) Cardiac procedures.

Evaluation

Evaluation is difficult and somewhat inaccurate but is carried out in four ways: (1) by use of the films as outlined above; (2) a final examination which is filmed, and consists of the performance of a standard operation (e.g. an end-to-end anastomosis of the jejunum); (3) examination of the laboratory manual; and (4) the recorded observations of the instructors and recorded personal communication with the candidates.

Comments

Without exception all students were more confident about surgical procedures at the end of the course. Improvement in technique, as judged by the crude methods of evaluation noted above, occurred in all students. The greatest improvement took place in the first two or three weeks of the course with another peak of improvement occurring near the end of the 12-week period. A basic minimal standard can be achieved by all students. Some students excelled, actually being able to perform a meticulous gastrectomy in 60 to 70 minutes. Many of these students were initially more dexterous than their fellow students, were "surgically motivated" and spent more time perfecting their technique.

Almost without exception the learning process is best carried out by having the student perform the procedure himself aided by comments and actual assistance from the instructor, and is not greatly advanced by demonstration operations.

The students who had taken the course proceeded to their surgical rotations during internship with greater confidence. There was some indication that a slightly greater proportion of these particular students were attracted to surgery as a specialty than their counterparts who did not take the course.

The course has given the instructors an opportunity to observe the essential features involved in the teaching-learning of surgical technique.

Résumé

Depuis huit ans, la faculté de médecine de l'université d'Alberta à Edmonton offre un cours facultatif de technique chirurgicale d'une durée de 12 semaines aux étudiants non diplômés du cours de médecine. Quelque 50 étudiants des première, deuxième et troisième années ont suivi ce cours, donné pendant les vacances estivales une fois par semaine à l'Institut de recherches médico-chirurgicales. Toutes les opérations se font sur des chiens. Le but de ce cours est triple: (1) rompre les étudiants aux techniques chirurgicales; (2) faciliter aux étudiants une poursuite plus efficace de leur programme de recherches estivales et (3) évaluer la façon dont les étudiants apprennent cette technique.

Il ressort de cette expérience que tous les étudiants avaient acquis ainsi une plus grande confiance dans la chirurgie. Cette amélioration était visible chez tous et se manifestait surtout durant les deux ou trois premières semaines du cours, les sujets retrouvaient un nouvel enthousiasme à la fin du cours. Tous les étudiants peuvent retirer de cet enseignement un bénéfice standard minimum.

Ce cours a par ailleurs permis aux enseignants de noter les caractéristiques pédagogiques que doit avoir cet enseignement spécialisé.

TOTAL VERSUS SUBTOTAL EXCISION OF ACOUSTIC NEURINOMA

With progress in anesthesia and the preoperative and postoperative management of neurosurgical patients, total excision of acoustic nerve tumours has become more popular, even though the technical difficulties and the traumatic effects remain.

The author stresses that total removal of the tumour requires great experience and high technical skill, especially if patients are elderly. It is for this reason and because of damage to the facial nerves, which can be of such serious consequences especially to young women, that the author recommends that anybody who has not yet fully developed his technical skills should use the less traumatic and yet sufficiently effective subtotal excision of the nerve tumours, which he described as early as 1939.

Of 17 patients operated on by this method only one died. Facial paresis of facial nerves was caused in two patients with far advanced tumours. None had evidence of recurrence three years after the operation.—Babchin, I. S.: Total versus subtotal excision of acoustic neurinoma, *Voprosyi Neirokhirurgii*, **2**: 1, 1969.

TREATMENT OF ORAL CARCINOMA WITH SELECTIVE THERAPY FOR THE INDIVIDUAL PATIENT*

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SQUAMOUS cell carcinoma of the lower oral cavity, i.e. the anterior two-thirds of the tongue, floor of the mouth and alveolus, is of such seriousness that nearly two-thirds of patients in whom it develops die of the disease. Moreover, successful treatment may produce significant cosmetic and functional disability. The aims in treatment, therefore, must be to produce the best cosmetic and functional result possible as well as to increase the number of patients cured. The method of treatment may be radiation therapy alone, operation alone, or a combination of both. From reports in the literature it appears that many centres select as treatment either operation or radiation almost to the exclusion of the other, and infrequently use the two in combination.[1-4] Yet there appears to be very little statistical difference in the cure rates obtained in these centres. Perhaps some radiotherapists have failed to realize that selected patients would do better when treated surgically, and undoubtedly those who state that all these patients should be treated surgically create unnecessary cosmetic and functional disability.

MATERIAL AND METHODS

In the years 1953 to 1962 inclusive, 158 patients with carcinoma of the floor of the oral cavity were seen at the British Columbia Cancer Institute (B.C.C.I.). The Manchester method of staging was applied to all patients before the start of therapy. The Manchester method of staging is as follows:

Stage I—Primary mobile, confined to the tissue of origin, less than 3 cm. in diameter, no nodes.

Stage II—Primary less than 4 cm. in diameter.

Stage III—Primary as in Stages I and II but cervical nodes on the side of the primary clinically involved.

Stage IV—Extensive primary; bilateral nodes, evidence of extracapsular spread or remote metastasis.

The majority of these patients were seen at a special clinic for tumours of the head and neck before treatment was begun. The clinicians attending the clinic included radiotherapists, specialists in head and neck surgery, and plastic and oral surgeons. The nucleus of the attending group of clinicians remained quite constant over the 10-year period. Each patient was individually assessed by the clinic members and the best method of treatment was then decided upon. When treatment had been completed these patients were all carefully followed by the same clinicians. If there was evidence of recurrence or metastatic disease, further treatment was determined by the group. In addition, clinicians are sent by the British Columbia Cancer Institute, at regular intervals each year, to maintain follow-up of patients living in the more remote areas of British Columbia. Very few patients, therefore, are lost to follow-up and in this particular study the follow-up was complete. In this group-clinic setting, the radiotherapist and the surgeon both acquired an increased appreciation of what the other had to offer in the treatment of these patients.

Included in this series of 158 patients were 10 who received no treatment of any

TABLE I.—SQUAMOUS CELL CARCINOMA OF THE LOWER ORAL CAVITY. BRITISH COLUMBIA CANCER INSTITUTE, 1953-1962

Cancer of	*Stage*	*Number of patients*	*Total no. of cases*	*Five-year survival (%)*
Tongue	I and II	44		
	III	10	84	33
	IV	30		
Floor of the mouth	I and II	25		
	III	13	47	27
	IV	9		
Lower alveolus	I and II	13		
	III	4	27	41
	IV	10		

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type because the disease was far advanced or because the patient refused the recommended treatment. The records indicate that at least 23 additional patients were treated with hope of palliation only. In Table I the site of origin, the clinical stage at the time of diagnosis and the absolute five-year survival rate are set forth. Non-treated cases are included in the calculation of the five-year survival rate.

Table II provides a comparison of the absolute five-year survival rate in this series with those in other published series.

TABLE II.—Five-Year Survival

Series	*Tongue* (%)	*Floor of mouth* (%)	*Lower alveolus* (%)
B.C.C.I.	33	27	41
University of Pennsylvania (Royster *et al.*[7])	24	—	—
Michael Reese Hospital (Uhlmann and Weiner[4])	23	—	—
Johns Hopkins Hospital (Ward *et al.*[8])	35	—	—
Mayo Clinic (Simons, Masson and Beahrs[3])	—	—	44
Emory University (Wilkins and Vogler[9])	—	—	31
Memorial Hospital, New York[5]	—	36	—

In most of the latter, radiation therapy or surgery was used nearly exclusively. In some of the other series results are determinate and not absolute.[5] We believe, therefore, that because the results of treatment in the group of patients we present are equal to those reported in the literature, our approach to treatment is a satisfactory one.[6]

Treatment Policy

Of prime importance in the determination of the method of treatment of each individual patient was the clinical stage of the disease at the time treatment was begun. Manchester Stages I and II indicate a small primary lesion with no evidence of metastasis. In Stage III disease there is a small primary but clinical evidence of unilateral mobile lymph-node metastasis. In Stage IV the disease is advanced, but some patients can still be treated with hope of cure. Approximately one-half (51.8%) of these patients had either Stage III or Stage IV disease at the time of diagnosis. The treatment policy outlined in Table III has generally been followed in the treatment of all patients. Experience over the 10-year period led to some modification and we believe if the policy that evolved had been more strictly applied, especially in the early years of this series, the results would probably have been even better.

Most Stage I and Stage II lesions should be treated by radiation therapy. Needle implants are used for the tongue and a radium mould for the floor of the mouth and alveolus. Very small lesions of the tip of the tongue may be surgically excised. In Stage III disease the primary lesion can be treated by radiation therapy followed, after its conclusion, by immediate radical neck dissection. This is especially true for disease affecting the tongue. We recommend, however, a composite resection as the primary treatment in Stage III lesions of the lower alveolus and in those of the floor of the mouth when there is any suggestion of fixation to, or involvement of, the mandible. By the term "composite resection" we mean an operation where the primary lesion, with a wide margin of surrounding normal tissue, is resected in continuity with the adjacent mandible and a radical neck dissection on the same side. A temporary tracheostomy is always performed in conjunction with such a resection. Reconstruction of the mandibular defect, where indicated, may be done either primarily or at a later stage. We have learned that attempts at mandibular reconstruction when composite resection has been done following radiation therapy are usually unsuccessful.

TABLE III.—Treatment Policy

Stage	*Treatment*
I and II	Radiation therapy by needle or mould
III	(1) Radiation to primary (2) Immediate radical neck dissection of nodes (3) Consider composite resection especially for growth of the lower alveolus
IV	(1) Composite resection when feasible (2) External cobalt radiation for cure or palliation of non-resectable tumours

Careful prolonged follow-up of all cases.

In patients with Stage IV disease that is judged to be surgically resectable with hope of cure, a composite resection should be done. In patients with non-resectable tumours, external cobalt radiation is given for cure or palliation.

It is essential that all patients be seen at regular follow-up for the rest of their

lives. In several patients in this series prompt recognition that the primary method of treatment had not been successful or early detection of recurrent or metastatic disease permitted further treatment with five-year survival. We do not advise prophylactic cervical node dissection. We recommend that in patients whose primary carcinoma has been controlled by radiation therapy or local surgery and who later develop clinical evidence of metastatic disease in cervical lymph nodes, radical neck dissection be done immediately, without biopsy proof. For those patients in whom there has been failure of treatment or a recurrence following radiation, a composite resection is recommended if the growth is judged to be resectable. External cobalt therapy is given in a further attempt to control disease in those patients who have failed to respond to surgical treatment. We emphasize that the choice in these patients is not between a major surgical procedure and a few innocuous radiation treatments. There is potential hazard to the patient by both methods of treatment and unless radiation therapy is given with skill and care there can be unquestionable patient morbidity. We recommend, for example, extraction of carious teeth with careful mucosal closure by specially trained dentists before radiation therapy is begun in order to reduce the hazard of septic complications and later radiation necrosis of bone. If patients who have had radiation therapy later require dental extractions, these also should be done by specially trained dentists, and antibiotic coverage is advisable.

TABLE IV.—Primary Method of Treatment in 148 Cases

Treatment	*Number of patients*	*Percentage of total*
Radiation to primary (subsequent radical neck dissection in 20)	102	69
Radiation to primary and immediate radical neck dissection	21	14
Local operation only	6	
Composite resection	19	17

Results of Treatment

In Table IV, the results of the primary method of treatment of the 148 patients, including 23 with hope of palliation only, are analyzed. Radiation therapy was the initial mode of treatment in 69% of patients. In 31% of patients, operation alone or in combination with radiation was selected.

The initial treatment according to the primary site of the disease, with the addition of radical neck dissection performed either in conjunction with the initial treatment or during the follow-up period, is indicated in Table V.

TABLE V.—Primary Method of Treatment Including Follow-Up Radical Neck Dissection

Cancer of	*Radiation +*	*Radical neck dissection*	*Composite resection*	*Local excision*
Tongue	68	26	5	5
Floor of the mouth	38	11	8	—
Lower alveolus	17	4	6	1

It is evident that composite resection was used more frequently in the treatment of carcinoma of the lower alveolus and floor of the mouth than of carcinoma of the anterior two-thirds of the tongue. Radical neck dissection played an important role in the management of patients whose primary tumour was treated by radiation. Forty-one of 123 such patients had an associated radical neck dissection with an almost even distribution of dissection immediately following radiation therapy and dissection done later in the follow-up period.

The analysis of this group indicates that the final die is not necessarily cast when the choice of treatment is first made. Again it is necessary to emphasize the importance of regular and careful follow-up so that if further treatment is needed it can be undertaken as early as possible.

TABLE VI.—Composite Resection Following Failure of Radiation*

Cancer of	*Number of patients*	*No palliation*	*Good palliation or cure*
Tongue	9	1	8
Floor of the mouth	2	1	1
Lower alveolus	2	0	2
	13	2	11

*There were six five-year survivors.

Table VI shows the use of composite resection following failure of radiation therapy in control of the disease and Table VII shows the use of radiation therapy following failure of composite resection.

Composite resection was performed in

the follow-up period on 13 patients in whom radiation therapy had failed to control the disease. Six of these 13 patients were five-year survivors and five others received good palliation. The salvage rate from radiation therapy following failure of a radical operation was not as good, although two of eight patients treated survived for five years and two others received good palliation.

TABLE VII.—Radiation Therapy Following Failure of Composite Resection*

Cancer of	*Number of patients*	*No palliation*	*Good palliation or cure*
Tongue.....	5	3	2
Floor of the mouth	1	1	0
Lower alveolus...	2	0	2
	8	4	4

*There were two five-year survivors.

Summary

A series of 158 patients with carcinoma of the lower oral cavity, seen at the British Columbia Cancer Institute during the years 1953 to 1962, has been reviewed. We believe that selective therapy for individual patients using either radiation therapy, operation, or radiation therapy and operation combined, when the indications for each method are seriously considered, has given as good a cure and palliation rate as is obtained in other centres, and by the careful selection of patients cosmetic and functional disability can be reduced. Careful follow-up of all patients is essential and when recurrence or metastatic disease is recognized early, further treatment can still result in cure. Cooperation between the radiotherapist and the surgeon is essential in this scheme of treatment. The criteria for case selection of the individual patients have been outlined.

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Résumé

Les auteurs présentent les résultats du traitement de 158 cas consécutifs de cancer de la cavité buccale qui ont été traités au British Columbia Cancer Institute. Ces cancers siégeaient aux deux-tiers antérieurs de la langue, au plancher de la bouche ou à l'alvéole. Ces résultats sont semblables à ceux qui ont été rapportés chez d'autres groupes de malades. Les auteurs soutiennent que, dans les cas où l'on dispose d'une radiothérapie adéquate et de chirurgiens compétents, on devra adapter aux besoins individuels de chaque cas le mode de traitement initial, selon la phase de la maladie. Dans les lésions localisées, le meilleur traitement est la radiothérapie. Il est souvent possible de guérir un cancer par ce moyen sans créer de cicatrices inesthétiques et sans entraîner d'invalidité fonctionnelle. Par contre, dans les cas plus avancés mais néanmoins curables, la chirurgie apporte le meilleur espoir de guérison. Une longue et attentive période d'observation post-thérapeutique est indispensable. Il importe de diagnostiquer précocement les récidives ou les métastases si l'on veut qu'un nouveau traitement aît des chances de réussir.

THE SKIN-GRAFTED BELOW-KNEE STUMP: CAN KNEE FUNCTION BE SALVAGED?

H. G. THOMSON, M.D., M.S., F.R.C.S.[C]* and D. BOCHMANN, C.P.O.[C],† *Toronto, Ont.*

THE below-knee (B-K) prosthesis with a "slip-socket" has been in use for several years and was designed primarily for the patient with a short B-K stump.[1-3] However, its specific application to the patient with a skin-grafted B-K stump has not been reported.

Slocum[4] and Speed and Knight[5] noted that skin grafts are impracticable in the region of total contact, weight-bearing stumps. It has been an accepted practice to excise skin grafts in these areas as a secondary procedure, then to apply traction and close the stump with healthy local skin before fabricating the prosthesis. However, in the case of a traumatic, short B-K amputation it is not always possible to obtain healthy skin and fat, either by local or distant means, for stump closure.

It is sometimes necessary to obtain primary wound closure with a split skin graft if the surgeon is to fulfil one of his major obligations, namely to preserve knee function. To do this, other means than the accepted new types of B-K prostheses must be employed.

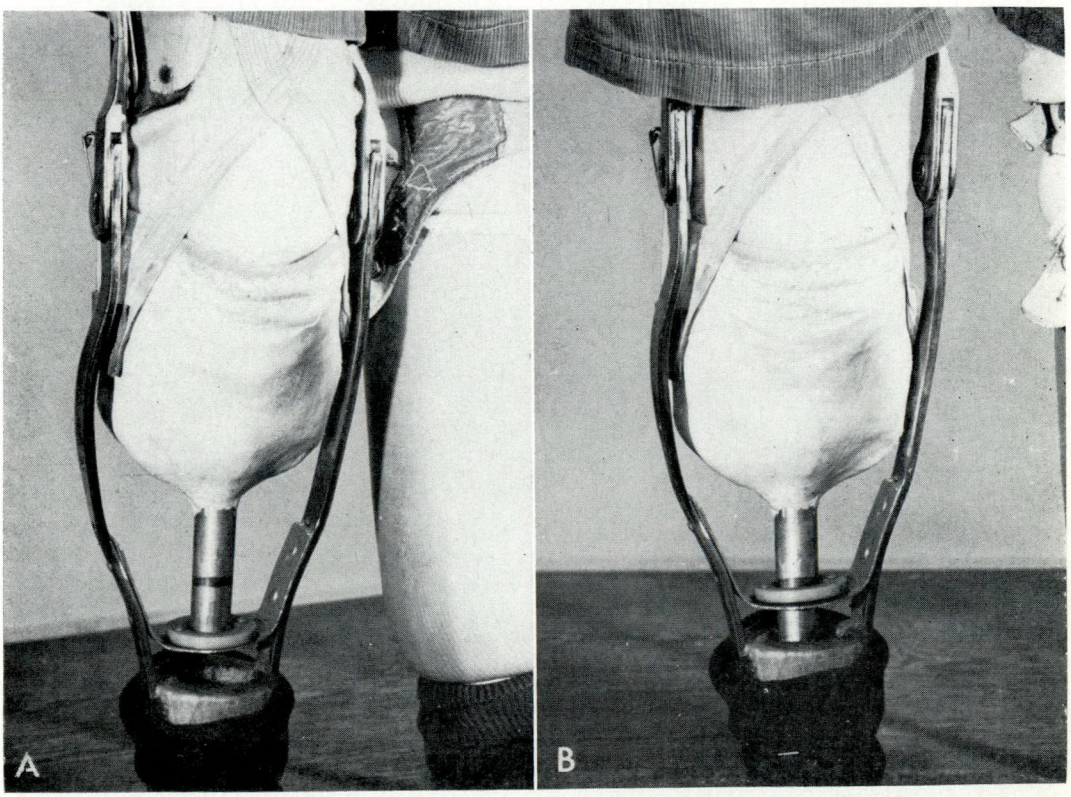

Fig. 1.—(A) Early postoperative prosthesis showing the position of the guide tube in the non-weight-bearing position—note the clearance allowed for the slip-socket. (B) Early postoperative prosthesis during weight bearing.

It is our purpose to acquaint surgeons with a practical type of prosthesis that can be fabricated when this unusual traumatic situation arises, a prosthesis that can possibly save the knee joint.

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Principle

The immediate skin-graft area on a B-K stump is usually fragile and susceptible to breakdown due to friction. The same is true, to a lesser degree, of the skin pedicle-flap graft. As the vascular supply becomes well established, and the graft becomes soft, mobile and mature, the likelihood of friction ulceration and maceration lessens. In a dependent B-K stump the skin graft may take as long as 8 to 12 months to mature. However, complete isolation of the skin-graft site does not spontaneously lead to maturation and a compromise, which permits slight friction and contact, is desirable. This type of protection cannot be provided by either the stationary patellar tendon bearing (PTB) or patellar tendon supercondylar (PTS) type of prosthesis or by the addition of a thigh corset with knee hinges.

It is necessary to use the skin-grafted stump with its good knee movement as a lever arm to activate the shank of the prosthesis. Therefore, the latter must have a free floating B-K socket which allows the stump to bear only a minimal amount of weight and also permits activation of the shank. This effect is achieved by connecting the socket to the lower shank in such a way that it will "rotate" and "piston" up and down as well as allow the knee joint to activate the shank of the prosthesis (Fig. 1).

The rest of the prosthesis is basically a quadrilateral socket with an ischial shelf. The contour-fitting stump socket is suspended from the quadrilateral socket by means of elastic straps. The knee hinges and distal leg are attached to the quadrilateral socket in the usual fashion.[6] It is made so that it can be adjusted as the stump shrinks.

The prosthesis is applied soon after operation. As the soft tissues of the stump become stable, the prosthesis can be altered to provide a pleasing cosmetic appearance (Fig. 2). This prosthesis may be converted to the standard PTB or PTS after skin-graft maturation has occurred.

Clinical Application

The Ontario Crippled Children's Centre (O.C.C.C.), with an enrolment of 589 patients, has one of the largest children's amputee clinics in North America. In this large group of patients there were only 13 with split skin grafts involving the area of contact with the prosthesis and nine others with some type of pedicle-flap graft to the lower limb. In this group of 22 amputees, only three patients required skin-graft coverage of the short stump in order to preserve knee function. They were treated primarily with the slip-socket prosthesis to ensure early ambulation, continued health of the skin graft and preservation of knee function (Fig. 3).

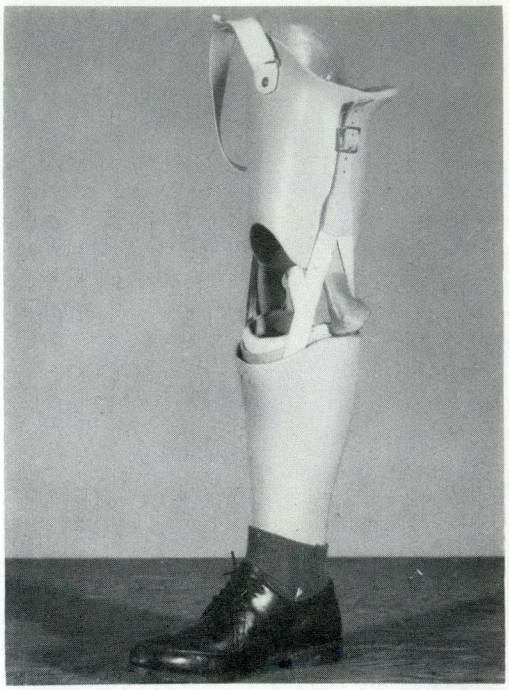

Fig. 2.—Permanent prosthesis showing similar adjustments but cosmetically pleasing.

All three patients had been involved in train accidents and were bilateral amputees. Their average age was 8 years. All six stumps required skin substitution in the form of pedicle or split skin grafts to the distal portion of the stump and/or the anterior surface and the tip. In two patients the slip-socket was fabricated within six weeks of discharge from the referring hospital. The other patient did not receive his first slip-socket until four months after initial discharge from the referring hospital, because of necessary revisions to the

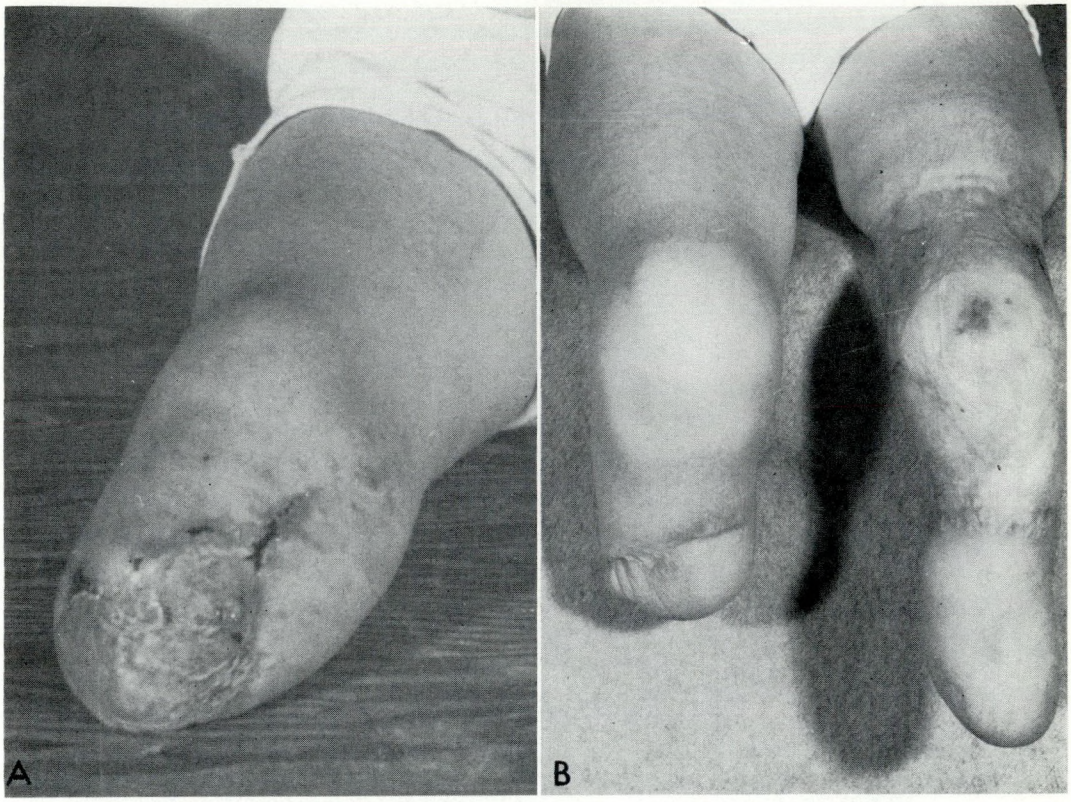

Fig. 3.—(A) Distal end of the stump covered with a split skin graft. (B) Another patient with the left stump covered by a distal pedicle-flap graft and a proximal free skin graft.

opposite stump. All patients used this type of socket for at least two years. In only one patient has there been conversion to a PTS prosthesis with high condylar flares and he is doing well.

One further patient, a bilateral B-K amputee with distal skin-graft coverage only, has recently been fitted with a slip-socket. Initially he had been fitted with a PTB prosthesis with thigh corsets, but there had been continued breakdown of the skin-graft sites on one side. After being fitted with a slip-socket he has been able to walk without harm to the grafts.

Discussion

Obviously the clinical situation necessitating this type of prosthetic fitting is rare. Indeed, one might wonder why so much attention should be given to such an insignificant point as saving the knee joint. It would be much easier to complete a higher amputation initially, to secure primary healthy skin coverage immediately after the accident, and allow early fitting of a prosthesis and rehabilitation. But to those familiar with the mechanical knee, even when it is supplied with the new polycentric hinge, it is obvious that this device cannot simulate the patient's own knee action and produce a smooth gait. One must repeat, therefore, that the surgeon should go to all reasonable lengths to preserve the patient's knee joint.

Since skin-graft maturation is a slow process, even in the child, patience is required on the part of the prosthetic team before the decision is taken to convert the patient to a standard PTB or PTS type of prosthesis. With the slip-socket technique, any impatience can be overcome through early ambulation, training and a reduction in the postoperative morbidity. This device may be a valuable addition to the surgeon's armamentarium when he attempts to rehabilitate his amputees.

SUMMARY

The use of the slip-socket principle as applied to children with B-K skin-grafted stumps is described. The clinical course of three patients is presented to demonstrate the advantages of this technique. This type of prosthesis allows early ambulation and preserves knee function, thus improving the quality of gait and increasing the capacity for activity by the amputee.

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RÉSUMÉ

Les auteurs décrivent l'application du principe du "slip socket" aux enfants amputés au-dessous du genou et qui ont une greffe cutanée. Ils illustrent cette technique en relatant l'évolution clinique de trois enfants. Ce type de prothèse permet une marche précoce et préserve la fonction du genou, améliorant par le fait même la qualité de la démarche et augmentant la capacité d'activité de l'amputé.

IMMEDIATE POSTSURGICAL FITTING OF THE LOWER EXTREMITY AMPUTEE

Experience with immediate postoperative application of a prosthesis after amputation in the geriatric patient has established certain operative techniques and regimens of postoperative care. Before this experience, amputations were performed with little hope of ultimate use of a prosthesis and of ambulation. At first the results were poor. Most of the patients had some complication. The most common complication was necrosis in the anterior flap. Meticulous care in surgical technique and the medical condition of the patient gave better results.

Myoplastic procedures were performed. These entail the attachment and covering of the bone end by muscle flaps which are tailored to create a smooth cylindrical stump or preferably a conical stump. Myeloplasty gives stability and movement to the stump and preserves some proprioception with muscle tension. Long posterior flaps where vascular supply is better have been created in preference to long anterior flaps.

Plaster cast is applied over a contoured dressing and sterile stump sock. Elastic plaster is used, and pressure is applied from below upwards with no circular compression. A coupling device and pylon are applied and, if possible, the patient stands the next day. With some patients, there is ambulation in parallel bars without weight bearing on the amputation.

At 14 days the cast is removed and two new casts are immediately applied after the sutures have been removed. One is a model for a plastic socket for a temporary prosthesis; the other is applied in order to prevent swelling. The patient goes into the plastic socket within a few days on a permanent basis. This is attached to a pylon for weight bearing. Not more than 50% of the weight bearing is permitted until after six weeks have passed even if the patient can walk with no pain. Shrinkage of the stump occurs only with ambulation and therefore early ambulation is conducive to early fitting of the definitive prosthesis.

Great care in gait training is emphasized. The final prosthesis is tailor-made for the individual patient since each will establish his own degree of weight bearing and prosthesis need.—Russek, A. S.: Immediate postsurgical fitting of the lower extremity amputee, *Med. Clin. N. Amer.,* **53:** 665, 1969.

REVIEW ARTICLE

FAT EMBOLISM*

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ALTHOUGH Zenker described fat embolism at autopsy in 1862 and Bergman described this syndrome in a living patient in 1873, it is still regarded as a rarity.

Wilson and Salisbury,[1] who found only eight such patients among 1000 battle casualties including 119 fractures of long bones, underrated its frequency. The incidence quoted by Newman,[2] 5.6% of long-bone fractures, is more realistic but still, I believe, too low. Sevitt,[3] after reviewing 25 such cases from the Birmingham Accident Hospital, Birmingham, England, estimated that 45% of patients with multiple fractures subsequently had fat emboli and that 13.6% of them had cerebral embolism. These estimates parallel our experience at the Aberdeen Royal Infirmary. The incicidence of fat emboli quoted varies widely because the range of presenting symptoms is so wide and the condition is, in consequence, difficult to define.

Whitson[4] has denied that fat embolism causes severe symptoms or even that these emboli exist. He cites the extreme rarity of reported cases after elective bone operations, the presence of stainable accumulations of fat in a high proportion of autopsies after operations that did not involve bone, and even in conditions where there has been no operation, and concludes that the one factor common to the many alleged causes is anoxemia.

On the other hand, at autopsy, evidence of gross pulmonary fat embolism is frequently found. Robb-Smith[5] found such evidence in 30% of patients dying from accidents and calculated that in 70% of these patients the embolism contributed significantly to the fatal outcome. Watson[6] in an autopsy study of 38 patients many of whom were elderly and had upper femoral fractures, found that 19 had pulmonary fat embolism and eight had systemic embolism.

The positions taken by Whitson, on the one hand, and Robb-Smith and Watson on the other are the extremes, but I think every surgeon would concede that there is a clinically recognizable condition known as "fat embolism", although some would not apply that term to it.

ETIOLOGY

Fat embolism is seen mainly after fractures of the long bones, particularly those of the lower extremities. Within this generalization, there are several puzzling exceptions, e.g. the relative rarity of this complication following hip fractures in the elderly.

Although it develops mainly after fractures, fat embolism has been described in other conditions, e.g. in patients with bone contusion without fracture. Severe soft-tissue injury (individuals who have been "beaten up") has provided some cases, and it can be produced experimentally by soft-tissue contusion. Fat embolism is frequently found at autopsy in patients dying of acute pancreatitis.[6] Moreover, I have seen a patient with a ruptured pancreas recover completely after coma lasting several weeks; I believe that fat embolism may have contributed to the coma. Caisson disease, diabetes, sickle cell anemia and alcoholism[7] have added their quota. Childbirth and thermal burns, on rare occasion, set the stage for this complication. Fat embolism is not infrequent after open-heart operations and has been ascribed to the use of the pump oxygenator. In the most recently described cause—prolonged high dosage of corticosteroids used to prevent rejection of kidney transplants—the fatty liver was thought to be the source of fat. Jones, Engelman and Najarian[8] described four patients who had undergone renal transplantation in whom the embolism developed immediately after the steroid had been reduced or stopped sud-

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denly. In one such death, the steroid dosage had been as high as 120 mg. daily; this patient had a fatty liver, petechial hemorrhages in the skin, gut and brain stem, and gross fat embolism in the lungs. Two of Jones' patients who survived developed avascular necrosis of the femoral head.

Fatal fat embolism has also been described in patients with rheumatic fever who developed steroid-induced Cushing's disease during treatment.[9]

With the exception of acute pancreatitis, most of these causes are rare—one might even say exotic—and, clinically, fat embolism is almost entirely a complication of fractures of the lower limb bones and especially multiple fractures.

Genesis of Fat Emboli

Possible Sources: Considerations of Pathology and Abnormal Physiology

The exact genesis of fat embolism is controversial. Gauss' explanation that the fat came from medullary sinusoids held rigidly open within bony trabeculae, was challenged by Lehman and Moore,[10] who believed that fat globules could be formed in the blood from the coalescence of chylomicra. *In vitro* they demonstrated that large-globule fat emulsions could be broken down by histamine, but could not show, either *in vivo* or *in vitro*, that fat emulsion in lipemic serum could be broken down by this means. Lehman and Moore calculated that the volume of embolic fat in the lungs was greater than the fat content of the fractured bone, and that the whole femur did not contain enough fat to produce fatal fat embolism, even if all of it entered the blood stream. Subsequently, Peltier[11] demonstrated that the amount of fat in the femur was far greater than Lehman and Moore's estimation and drew attention to the high fat content of the metaphyseal region. It must be remembered, also, that there is a wide species variation in the degree of susceptibility to fat embolism, the dog is more liable than the rabbit, and the rabbit is more liable than the rat. Perhaps man is peculiarly susceptible.

Krönke[12] found that the fractured tibia in a case of fat embolism contained 0.5 g. more fat than the uninjured bone. However, anyone who has seen liquid fat running out of a femur after osteotomy will find this hard to accept unless fat is lost from both femurs.

More recently Bergentz[13] has strongly supported the concept of intravascular formation of fat globules. In rabbits he demonstrated that fat embolism occurred within two hours of injury, that it was associated with migration of triglycerides from the plasma to the red cell mass, and that later the viscosity of the blood increased when high-molecular-weight fibrinogen and globulin produced intravascular aggregation of red cells and a decreased suspension stability. He thought that this series of changes caused fat globules to form, that any injury could produce fat embolism and that fractures are significant only because they represent severe injury. The globules that he produced were, however, rather small, up to 12μ, whereas the pulmonary emboli in the clinical syndrome are 50 or even 100μ. Bergentz, in fact, views thrombus formation and fat embolism as part of the same process. Adkins, Foster and Saile[14] have produced fat embolism by injecting thrombin and showed that fasting but not lipemic animals could be protected by heparin.

Fuchsig *et al.*[15] demonstrated that, in rabbits with a reduced blood volume, ^{131}I-labelled triolein was rapidly absorbed from the subcutaneous tissues, but after the blood volume had been restored, its absorption ceased. They concluded that, irrespective of its etiology, shock rapidly flushed out fat that was not fixed in a cell system. They thought that this fat found its way into the blood stream via the lymphatics, but Szabó, Serényi and Kocsar[16] have since shown that its route to the lung is primarily venous.

In the past, surgeons have been unwilling to accept the idea that marrow fat embolizes because they supposed that it was difficult for marrow fat to enter ruptured venous sinuses. However, Young and Griffith,[17] working in the Departments of Pathology and Medical Physics in Aberdeen, showed that where emboli could form, blood vessels with various intra-

vascular and extravascular pressures did not collapse completely but were in a state of unstable equilibrium, alternately opening and closing, thus permitting emboli to enter.

Because the total amount of plasma fat depends so much on alimentary lipemia, it serves no diagnostic purpose to estimate the total amount of blood fat. The demonstration of large fat globules in the blood as described by Peltier[18] is, however, significant.

In the rat, Morton and Kendall[19] demonstrated a marked increase in the number of fat globules in the femoral vein on the side of the fracture compared with the contralateral side. This important experiment proves that fat globules do originate at the site of injury.

Sevitt[20] strongly supports the concept of marrow fat embolism. In autopsies on accident cases, he has demonstrated recognizable bone-marrow emboli lying alongside large fat emboli; this most cogent argument has been confirmed by many others. Thus it has been clearly established, both clinically and experimentally, that fat embolism can originate at the site of injury. Nevertheless, we must also accept that the products of injury other than spilt marrow fat can produce the aggregation of fat droplets. In my opinion each mechanism is compatible with the other and I see no reason why both should not operate together in the same patient.

Because blood vessels in bone are under autonomic control, blood flow through them can be reduced by sympathetic stimulation. Bloomenthal, Olson and Necheles[21] showed that the intramedullary pressure, normally mid-way between the arterial and the venous, falls after adrenaline is injected, proving that a hormonal control operates as well. He also demonstrated that the intramedullary pressure in one femur fell after the contralateral femur was fractured; all the while the arterial blood pressure was maintained at normal levels.

The Role of Hypovolemia

The consideration of Bloomenthal's work emboldens me to propound my own concept of fat embolism. It would be extraordinary if the blood flow through bone, estimated in experimental animals to be between 5% and 10% of the cardiac output in the resting condition,[22] were not altered in hypovolemic shock. At the time of injury blood flow through bone is not essential to the individual's survival, and thus we would expect bone to participate in mechanisms to restore normal blood volume. In the average fracture of the femoral shaft, the circulating blood volume is reduced by about 1.5 1. This may be expected to produce a shut-down of intramedullary circulation that contributes to the perfusion of more vital areas. Its duration depends on the speed with which normal blood volume is restored. Until the blood volume was restored marrow fat, blood and products of injury would stagnate at the fracture site and, possibly, the longer the period of stagnation, the more toxic the product. When normal blood volume is restored and the medullary circulation reopens fat and the toxic products of injury probably pass rapidly into the blood stream.

To prove this theory we would have to monitor continuously the intramedullary pressure in the first 48 hours after a long-bone fracture in man, and do an intermittent quantitative count of the numbers of large fat globules. So far this has not been possible.

If one concedes that fat can enter the blood stream from the marrow cavity, and that fat emboli may form in the local venous circulation, pulmonary embolism of some degree is then automatic. Watson,[6] to explain the apparent difficulty that large emboli have in passing through the lung capillaries, proposed the following hypothesis. Fat emboli, although fluid at body temperature, have a viscosity greater than plasma, and therefore accumulate in the lung capillaries and increase the peripheral resistance. Secondarily, the pressure in the pulmonary artery rises until it is high enough to force emboli through the capillary network. A shower of emboli thus enters the systemic circulation, the pulmonary artery pressure falls and the cycle is repeated. This sequence accords with the clinical observation of periodic showers

of petechiae and for it one need not postulate a patent foramen ovale.

Once in the systemic circulation, emboli are distributed to the skin, the gut and every organ in the body, and lodge there or pass through according to the characteristics of the local vessels. If they lodge, microinfarcts form with consequences that are most marked in the brain. Fat emboli are more numerous in the gray than in the white matter but, because of the profuse capillary network in the gray matter, petechiae are seen only in the white matter. These petechiae are due to the blocking of end vessels. It is curious that embolization is particularly heavy in the posterior lobe of the pituitary—to a degree, according to Sevitt, exceeded only in the kidney. Emboli in the anterior lobe of the pituitary, however, are infrequent because this part of the gland has no direct blood supply. The extent and the precise location of cerebral emboli determine the outcome.

Elimination of Fat Emboli

The amount of fat excreted by the kidney or appearing in the sputum is insignificant. Fat is eliminated by two main mechanisms: first, the peripheral action of lipase and second, certain metabolic processes within the liver. In this respect the action of blood lipase, which is elevated in patients with fat embolism, is probably of prime importance. For the most part fat emboli do not pass through the liver sinusoids although fatty infiltration of liver cells is often found in fat embolism. Lipolysis is probably less effective in the fatty liver of the alcoholic, an individual who is at special risk from fat embolism after fracture. Emboli are broken down to fatty acids and glycerol. Although the fatty acids are quickly bound to protein it is possible that locally released fatty acids may increase lung edema and rigidity. Peltier clearly demonstrated the local toxic effect in the lung of unsaturated fatty acids.

Because edema of the lung can be produced in experimental animals by injecting fat into the carotid artery, some have deduced from this that such edema is secondary to cerebral embolism. Nevertheless, local blockage by fat of capillaries in the lung must be an alternative or additional cause of edema.

Clinical Features of Fat Embolism

Clinically we consider patients with fat embolism as belonging to one of two groups, the pulmonary or the cerebral; this division, however, is artificial. Every patient with a cerebral embolus must have pulmonary emboli. Indeed, Sevitt considers that the most severe pulmonary symptoms are produced by cerebral emboli. Nevertheless, in some cases the pulmonary symptoms, however mediated, are most marked. Such patients are cyanosed, have an increased respiratory rate, a fast pulse, an elevated temperature and look as if they have pneumonia.

Radiologically the lung parenchyma in established pulmonary fat embolism resembles a snow-storm because of multiple peribronchial fluffy infiltrations that are usually localized to one or both lower lobes. Clinically these features are usually explained in terms of congestive edema and patchy atelectasis.[3]

The symptoms of cerebral dysfunction, by far the most dramatic in this syndrome, dominate the clinical picture. The small areas of cortex "knocked out" by the occlusion of end-arteries are not in themselves as significant as the anoxia produced both by the pulmonary changes and the central nervous system depression. This point is illustrated by the following patients.

Case 1.—A 28-year-old man, the driver of a crashed car, was admitted to hospital with fractures of the shaft of the femur, humerus and patella, and rupture of the liver and duodenum. He was fully conscious and in shock—his blood pressure was 74/40 mm. Hg. After receiving four pints of blood and appropriate surgical treatment his condition was good and remained so for the next 24 hours. He then quite suddenly became cyanosed and stuporous. The house surgeon, thinking that this man might have a subdural hematoma, sought a neurosurgical opinion. The patient, however, had many petechiae on his chest and a few in his conjunctiva, and was clearly suffering from fat embolism. His Po_2 was only 48 mm. Hg. Undoubtedly he had cere-

bral emboli and his cerebral status was being aggravated by anoxia; from the combined effects of embolism and anoxia he was rapidly slipping into coma. Oxygen given by mask raised the Po_2 to 64 mm. Hg and subsequently, after tracheostomy and ventilation, it rose to 82 mm. Hg. Within an hour or two he was quite sensible.

This patient illustrates well the interdependence of pulmonary and cerebral symptoms. Without adequate oxygenation he would have gone into a descending spiral due to cerebral anoxia from multiple petechial hemorrhages and anoxemia.

Case 2.—This 29-year-old man was not so fortunate as Case 1. He sustained bilateral femoral fractures when his car somersaulted. The accident occurred in the country and five hours passed before he reached the operating room. When he was admitted he was fully conscious and had a blood pressure of 100/52 mm. Hg. After he had received five pints of blood his blood pressure rose to 135/90 mm. Hg, but by this time he was disorientated. After femoral traction was applied under anesthesia he recovered to the same confused level that had been present immediately before operation. Seven hours later he suddenly became cyanosed and began to produce frothy bloodstained sputum. He lapsed into coma and, in spite of tracheostomy, ventilation, hypothermia, Rheomacrodex (dextran 40), heparin, hydrocortisone and renal dialysis, he never recovered. He died 12 days later.

In this case there was considerable delay before the patient reached hospital and received adequate blood replacement. This delay undoubtedly contributed to the magnitude of his cerebral damage and its fatal outcome.

These two patients are typical of those with severe fat embolism, and between them lies the very narrow line separating survival and death. Most patients with disorders of consciousness short of coma will recover. The majority, but not all, of those who slip into coma will die. One exception was a man of 24 who was admitted deeply unconscious the day after he sustained a simple and only slightly displaced fracture of the tibia when his tractor capsized. He had petechiae in the conjunctiva, neck, chest and groin. An interesting point in his history was that on the day before the accident he had been ill with diarrhea and vomiting, and was presumably dehydrated. On the day after admission we could rouse him slightly, but it was four weeks before he became fully conscious. For the greater part of that time he lay apparently deeply asleep, opening his eyes occasionally to glare balefully at his surroundings. He made a complete recovery nevertheless. This is the only patient I have ever had who developed fat embolism twice, the second time 15 years later following a fracture of the pelvis. On this admission he had a blood pressure of 90/60 mm. Hg which we raised to 130/80 mm. Hg with three pints of blood. Forty-eight hours later he developed a crop of petechiae, but he never had cerebral symptoms to a degree that produced anxiety. (It is interesting to speculate whether there is any individual susceptibility. Certainly some people's bones have more fat than others.)

In the past I feared the prospect of having to graft a fractured tibia in a man who had severe cerebral symptoms from fat embolism after the original injury; however, on the one occasion on which this was necessary the patient passed through the postoperative period without complications.

In contradistinction to cerebral dysfunction producing coma, some patients become obstreperous, noisy and abusive. Their state may be confused with delirium tremens which can produce an identical mental state. Autopsy evidence of fat emboli in chronic alcoholics who die suddenly[7] suggests that delirium tremens may be due to emboli from a fatty liver.

One unfortunate patient, because she had a previous history of mental illness, was certified and sent to a mental hospital a week after she fractured her femur and pelvis. She was kept there until I rescued her three weeks later.

The interval between fracture and the onset of cerebral symptoms varies from a few hours to five days. The earlier the onset, the more severe the illness is likely to be. These patients often have slight spasticity and an extensor plantar response.

Urinary incontinence is common and may indicate a degree of cerebral disturbance. One of my patients who had a

severe head injury and a fracture of the tibia and fibula developed marked polydipsia and polyuria, but not until the fourth day after her fracture. These symptoms, which were controlled by Pitressin (beta-hypophamine), could have been due either to cerebral contusion or fat embolism; the late onset would seem to favour the latter. She still requires Pitressin three years later.

Petechiae

Although nearly every patient has petechiae the number of skin petechiae bears no relation to the severity of cerebral embolization. I have seen a patient die with only a few petechiae following bilateral femoral shaft fracture and, on the other hand, have seen several with petechiae extending from the root of the neck to the groin, whose mental disturbance never went beyond mild confusion. The reason, presumably, is that in severe shock the skin vessels are constricted to maintain the cerebral circulation, so that in severely traumatized patients the brain gets the lion's share of both the blood and the fat.

I cannot explain the typical distribution of petechiae to the volar aspect of the trunk and limbs.

As has been shown histologically petechiae are microinfarcts identical with those produced by the Hess test for capillary fragility. As far as I know this similarity was first noted by Sachs in 1951. He had made frequent blood-pressure recordings in a young man who had been unconscious for four weeks, and observed that many petechiae formed in the forearm after the cuff had been on for a very short time. Subsequently, using Scarborough's apparatus, he investigated capillary fragility using negative pressure over a small area so that he could repeat the observations on adjacent skin. By this method he found that when capillary fragility was normal he could produce only a few petechiae by a negative pressure of about 300 mm. Hg for half a minute. Possibly these petechiae are due to damage to capillary endothelium by fatty acids. Others have suggested that they may be produced when calcium is withdrawn to neutralize fatty acids. In this connection, Sproule, Brady and Gilbert[23] discovered that three patients with fat embolism had calcium levels between 7.8 and 8.6 mg./100 ml.; in one the embolism was fatal. Furthermore, after acute pancreatitis[24] patients sometimes develop tetany when they withdraw calcium to form soaps to neutralize the fatty acids released by fat necrosis.

Pyrexia

Pyrexia is invariable in fat embolism and occasionally these patients have hyperpyrexia. To date we have no good explanation of this pyrexia although some have blamed concurrent pneumonia, the action of toxins secondary to tissue damage and possibly the effect of anoxia on the fever centre in the brain stem.

Cardiac Changes

Tachycardia is also invariable. A sudden rise in the pulse rate to 120 or 130/min. in the absence of obvious cause, suggests that petechiae may follow. Peltier[18] describes typical electrocardiographic changes, namely a prominent S wave in lead I, prominent Q wave in lead III and inversion of the T wave. At autopsy myocardial and endocardial petechiae are frequent. Tachycardia is important in the differential diagnosis between fat embolism and intracranial hematoma. In the latter, the pulse rate slows.

Peptic Ulceration

An occasional, and potentially fatal, manifestation, gastrointestinal bleeding secondary to peptic ulceration, developed in a patient of mine, an innkeeper with osteoarthritic hips. Two days after he submitted to an intertrochanteric osteotomy he became delirious. In the absence of any obvious petechiae, his mental state was ascribed to delirium tremens. Twenty-four hours later he had massive hematemesis. Six pints of blood failed to raise his blood pressure. He lapsed into coma and died two days later. At autopsy he had several acute bleeding peptic ulcers and bilateral adrenal hemorrhage. The whole subperitoneal surface of the small bowel was shot through with petechiae. The liver was large and fatty. Not until a year later did I become aware that fat embolism could cause hematemesis. Harrison's[25] patient,

after he had symptoms of cerebral fat embolism, had hematemesis and recovered. Teare, Bowen and Drury[26] described three patients, one with massive hematemesis and two with perforated peptic ulcers. All had typical cerebral fat embolism and all died. Clearly, this peptic ulceration is a grave complication and one which, in a patient with already disturbed consciousness, is difficult to diagnose. When autopsies are done on patients dying from injury, 20% have peptic ulcers and Watson[6] found petechiae in the intestinal tract in 40% of fatal accident cases. Although embolism, with infarction of a mucous surface, is an obvious cause some authors attribute the ulceration to a hypothalamic effect caused by mid-brain emboli.

Possibly the most difficult patient to assess is the man with multiple fractures and a head injury, who is admitted unconscious, and who also develops typical petechiae. A burr-hole exploration may well be negative and the relative importance of the head injury and cerebral fat embolism may be impossible to weigh.

Treatment of Fat Embolism

The results of treatment of fat embolism are difficult to assess because the range of severity is so wide. For this reason it is meaningless to compare two series treated by different methods.

The various therapeutic measures fall into two categories—those that must surely be of value and those that may be.

Immobilization

There is, I maintain, a connection between the complication of fat embolism and delay in treatment. A patient with a fracture of the femur is always at risk—a risk that is increased if he has to travel far with the leg unsplinted. The patient who has to travel a long distance arrives in better condition if he has been immobilized in a Thomas splint than if he is not so immobilized. The difference is even more marked if both femurs are broken.

Blood Transfusion

As noted above I believe there is a connection between oligemia and fat embolism. If one accepts this one must transfuse early and adequately.

Oxygenation

When fat embolism is already established, oxygenation is of great value, with or without tracheostomy, particularly if the patient's condition is monitored by repeated blood-gas studies. Alone, supplementary oxygen can be expected to wake the patient who is semicomatose. We must maintain a high oxygen tension in alveolar air if we are to promote adequate gas exchange in a rigid lung and reduce the cerebral anoxia which must greatly exacerbate the local effects of cerebral petechiae. Of these measures, I would place oxygen saturation above all others. Many times[27, 28] patients have recovered from coma due to fat embolism after a tracheostomy and ventilation with oxygen. As in other causes of severe cerebral damage with coma, these measures have, with some success, been combined with chlorpromazine and surface cooling.[29]

Dextran

Bergentz[13] described a patient who was brought out of coma after receiving a bottle of dextran 40, lapsed into coma again, and was brought round a second time by a further litre which was repeated daily until the patient recovered completely. On such good authority I am prepared to believe that this measure is valuable, but obviously dextran will never be given alone, and hence its effectiveness will be difficult to assess.

Heparin

In the treatment of fat embolism, heparin has many supporters. Early reports of experimental work in animals were frightening[30] because it appeared to accelerate death. Sage and Tudor,[31] however, described three patients who recovered after small doses of heparin. However, I am reluctant to use any drugs that may increase hemorrhage.

Adler and Peltier[32] reported that lipase activity increased after they administered sublingual potassium heparin. This agent, they claim, has a fat-clearing property

without being an anticoagulant. Although I have no experience of its use, it appears to have promise. It would have to be given early because by the time the cerebral catastrophe has occurred it would be too late.

We once prescribed heparin intravenously for a man with severe fat embolism and because he was cyanosed put him in an oxygen tent. The next day he was greatly improved and we were prepared to give the credit to heparin until we found that, because of an error in communication, he had never received it. This salutary uncontrolled experiment has discouraged us from prescribing heparin.

Ethyl Alcohol

While heparin therapy is based on the premise that lipolysis is beneficial, intravenous ethyl alcohol, which depresses the action of lipase, is given on the premise that lipolysis is harmful. Since Peltier has shown that lipase acts at the point of embolization, these two methods of treatment are clearly incompatible. We do not advocate the use of alcohol.

Hydrocortisone

We do, however, believe that hydrocortisone is of some value because it may reduce the edema around cerebral petechiae. To all patients with cerebral signs, we give an initial dose of 200 mg. and then 100 mg. on two successive days before tapering the daily dose.

Unfortunately, many patients in deep coma still die despite the methods of treatment recently introduced. However, more will survive than formerly, and those that do survive will probably recover completely and have no residual disability due to fat embolism.

Summary

Fat embolism, a distinct clinical entity, is a frequent and potentially lethal complication of multiple fractures and many other forms of non-skeletal trauma. The overall incidence is undoubtedly higher than generally realized and many such cases probably go unrecognized. I have reviewed some of the significant studies into the pathogenesis of fat embolism and described the typical pathologic and clinical features, most of which are cerebral and pulmonary. Prevention is the best treatment, and fractures must be immobilized early and blood volume restored rapidly and completely by transfusion. Once the diagnosis of fat embolism has been made, adequate oxygenation is the most important single therapeutic effort.

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RÉSUMÉ

L'embolie graisseuse, entité clinique distincte, est une complication fréquente et potentiellement mortelle des fractures multiples et de nombreuses autres formes de traumatismes importants. Sa fréquence globale est incontestablement plus élevée qu'on ne le croit généralement et cette complication est trop souvent ignorée. L'auteur a passé en revue diverses études sur la pathogénie de l'embolie graisseuse et a décrit ses traits cliniques et pathologiques essentiels, dont la majorité porte sur des lésions cérébrales et pulmonaires. La prévention demeure le meilleur traitement et, pour ce faire, il importe d'immobiliser précocement les fractures et de rétablir rapidement et complètement la volhémie au moyen de transfusions. Une fois le diagnostic posé, le meilleur acte thérapeutique consiste à assurer une oxygénation convenable.

FAT EMBOLISM

The mechanical phase of fat embolism was demonstrated in rabbits by injecting neutral fat. Within two hours of fat injection, fat embolism produced symptoms similar to those causing death from respiratory failure, pulmonary hypertension, and heart failure on the right side. Histologic evidence of pulmonary as well as systemic embolism was seen in all rabbits. Serum lipase activity showed a graded rise after fat embolism occurred. Treatment with heparin was helpful, but sodium desoxycholic acid was ineffective. Definite proof of a chemical phase causing symptoms and pathologic lesions could not be clearly demonstrated. Almost all rabbits dying of pulmonary embolism had marked bronchospasm or vasospasm, or both, besides vascular occlusion by fat. This factor seems significant in causing sustained anoxia and subsequent fatalities.—Sachdeva, H. S. *et al.*: Fat embolism, experimental study—part I, *Amer. Surg.*, **35:** 250, 1969.

Adrenocorticotropic hormone and Phenergan are of benefit in experimentally produced fat embolism in rabbits. Smooth muscle relaxants were used in this study to evaluate their therapeutic effect on fat embolism. Adrenocorticotropic hormone and Phenergan produced the best results. They eliminated the respiratory and cardiac complications and prolonged the survival period. Adrenalin and dihydroergotamine produced some clinical amelioration of symptoms, but did not significantly prolong survival. Animals treated with atropine and papaverine showed less respiratory complications.

Death in fat embolism is caused partly by anoxia, resulting from mechanical blockage of pulmonary circulation caused by fat globules, and by the toxic effects of histamine-like substances liberated at the site of tissue injury. Drugs that counteract vasospasm and bronchospasm, and neutralize any histamine-like substances liberated by the injured tissues are of benefit.—Sachdeva, H. S., Sachdeva, K. C. and Chakravarti, R. N.: Fat embolism; experimental study—part II, evaluation of smooth muscle relaxants in its treatment, *Amer. Surg.*, **35:** 257, 1969.

CASE REPORTS

HEMANGIOPERICYTOMA OF THE SMALL BOWEL: CASE REPORT AND REVIEW OF THE LITERATURE

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In 1942 Stout and Murray[1] suggested the term "hemangiopericytoma" for a rare but distinctive soft-tissue vascular neoplasm. Since then this subject has been reviewed by Fisher[2] and O'Brien and Brasfield.[3]

The case presented in this paper is interesting because the tumour was located in the ileum and because it was associated with an ileocolic intussusception. In reviewing the literature we found that only three other cases of hemangiopericytoma of the ileum had been recorded,[4-6] and the only reported case of hemangiopericytoma of the jejunum was also associated with an intussusception.[7]

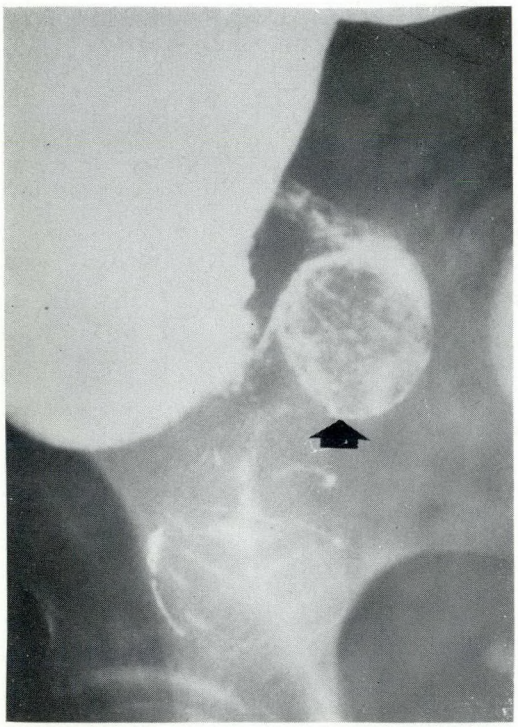

Fig. 1.—Barium enema. Reflux into the terminal ileum outlines the intraluminal tumour (arrow).

Fig. 2.—Repeat barium enema showing ileocolic intussusception. Subsequent films had a "coil spring" appearance.

Case Report

Mrs. S.K., a 72-year-old widow, was admitted to a local hospital complaining of a persistent cough productive of small amounts of mucus. She had been anorexic and had lost 16 lbs. in the previous two months. Her bowel movements had been regular and she had never noticed melena. While in hospital she had right upper quadrant abdominal pain which was aggravated by coughing and change of posture. A barium enema revealed an intraluminal tumour in the terminal ileum (Fig. 1).

She was transferred to University Hospital,

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Fig. 3.—Sessile, polypoid, intraluminal, focally ulcerated tumour of the terminal ileum. The surrounding mucosa is edematous and congested.

Saskatoon, for further investigation and treatment. A poorly defined tender mass was palpable in the right mid-abdomen, and a repeat barium enema showed an ileocolic intussusception (Fig. 2). On admission her hemoglobin was 14.8 g./100 ml.

Two days after admission her abdomen was explored and the diagnosis of ileocolic intussusception was confirmed. The intussusception was reduced and a segment of ileum about 13 cm. from the ileocecal valve, which contained the intraluminal tumour, was resected. End-to-end anastomosis was then performed. No metastatic lesions were found. The patient had an uneventful postoperative course and has continued well during the two years since her operation.

The resected specimen, which was 17 cm. long, contained a sessile, intraluminal, brownish-grey, polypoid lesion measuring 2.3 x 2.0 x 1.5 cm. which showed small foci of ulceration (Fig. 3). On cross-section the tumour consisted of firm, glistening, homogeneous, brownish-grey tissue extending down to the circular layer of the muscle coat. On microscopic examination this was a hemangiopericytoma. The tumour cells were stellate, polyhedral or elongated and possessed large vesicular ovoid nuclei (Fig. 4). Mitotic figures were not observed.

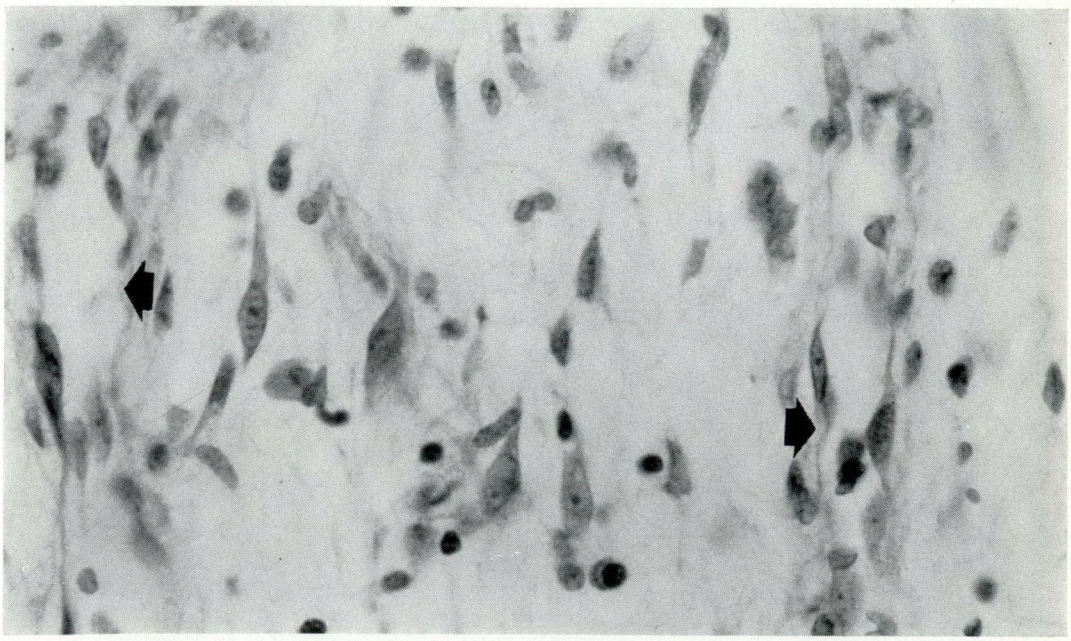

Fig. 4.—Blood vessels (arrows) are separated by elongated neoplastic cells which are widely spaced due to edema. A few lymphocytes are seen in the lower part of the field. In other areas the tumour cells were compactly arranged (H & E, original magnification × 850).

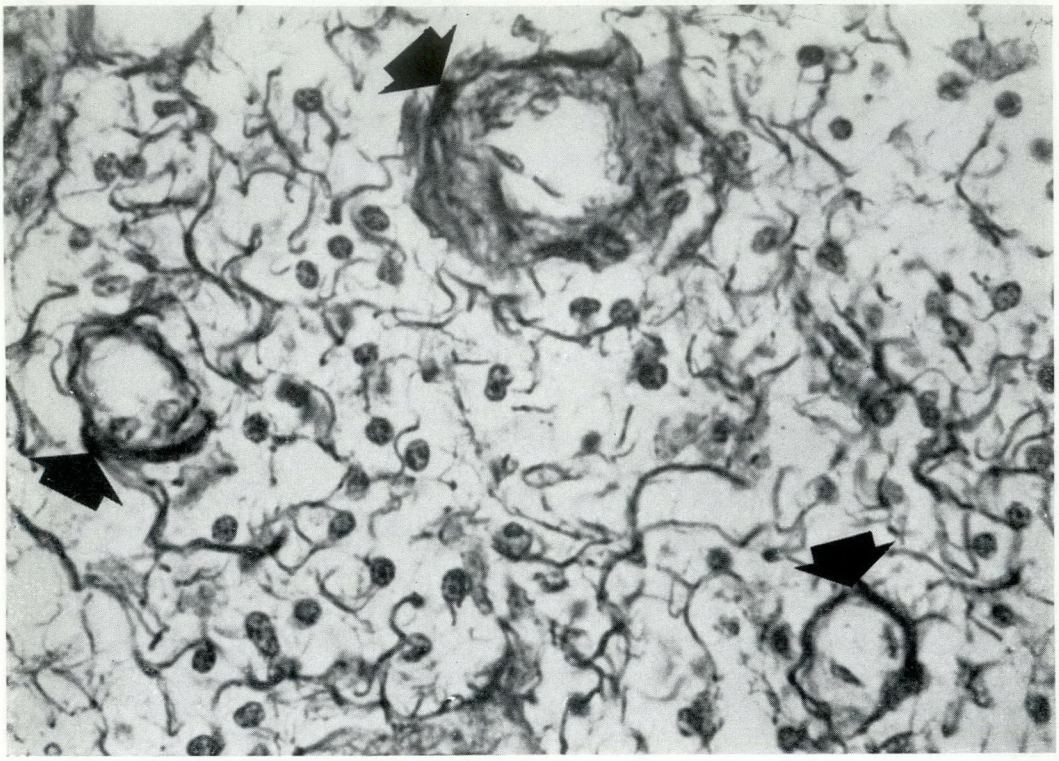

Fig. 5.—Reticulin fibres outline the blood vessels (arrows) and individual fibres surround the majority of the neoplastic cells (reticulin stain × 850).

Reticulin fibres outlined most of the neoplastic cells and demonstrated that they were outside the numerous vascular channels (Fig. 5). The base of the tumour was well preserved. Superficially it was ulcerated and the slightly edematous tissue contained inflammatory cells and hemosiderinophages.

Discussion

In a review of the literature we found only four additional cases of hemangiopericytoma of the small intestine: these cases and ours are summarized in Table I.

Although the follow-up on the reported cases is incomplete there is considerable evidence of the potentially lethal character

TABLE I.—Reported Cases of Hemangiopericytoma of Small Bowel

Author	*Age (years)*	*Sex*	*Presenting features*	*Site of tumour*	*Description of tumour*	*Operation*	*Clinical course*
1. Stout[4] (Case 23)	59	M	Paraumbilical, sharp, colicky pain	Lower ileum	4 cm. thick x 1.7 cm. wide, deeply ulcerating and constricting	Resection of lower ileum. Secondary tumour present in mesenteric lymph nodes at operation	Died 18 months after operation. Hepatic metastases present
2. Smith and Swenson[5]	43	F	Abdominal cramps, nausea, vomiting, abdominal distension	Terminal ileum	10 x 12 cm., subserosal	Enucleation of tumour	Not stated
3. Paulino and Torres[6]	56	M	Not stated. Developed anorexia, vomiting and weakness with recurrence of tumour	Terminal ileum	Original tumour the size of a nut. Recurrence was large	Resection of 15 cm. of ileum. Recurrent tumour treated by irradiation	Followed for 8 years. Recurrence with hepatic metastases at last follow-up
4. Thompson[7]	36	M	Crampy periumbilical pain, vomiting, distension, constipation	Jejunum	4 x 2.5 x 2 cm., mural, protruding intraluminally	Reduction of intussusception, jejunotomy and enucleation of tumour	No follow-up
5. Larsen and Cram (present case)	72	F	Anorexia, weight loss, right upper quadrant abdominal pain	Terminal ileum	2.3 x 2.0 x 1.5 cm., mural, protruding intraluminally	Reduction of intussusception and segmental resection of ileum	Well for 2 years

of the tumour: two patients developed metastases, one of these died after 18 months and, after eight years, the second was in a terminal state. Hemangiopericytomas may project into the lumen of the gut and may precipitate intussusception, may protrude as subserosal masses or may replace the bowel wall and ulcerate.

Segmental small-bowel resection and end-to-end anastomosis should be done on all small-bowel tumours where there is a clinical suspicion of malignancy. If the tumour is considered to be benign at operation, and excisional biopsy or enucleation is done, the lesion should, nevertheless, be submitted for diagnosis by frozen-section examination. Both surgeon and pathologist should be aware that hemangiopericytomas occur in this part of the intestine. If, after frozen section, the diagnosis of hemangiopericytoma is made or suspected, the surgeon should proceed with segmental small-bowel resection. He should also search for metastases at the time of operation. Finally, a prolonged follow-up is mandatory.

Summary

A 72-year-old woman had a hemangiopericytoma of the ileum. This lesion, which is rare in the small bowel, was associated with ileocolic intussusception.

Only four other cases of hemangiopericytoma of the small bowel were found in a review of the literature. The malignant potential of this tumour was manifested by the death of one patient 18 months after operation and the terminal state of another patient eight years after his original operation.

It is recommended that the management of hemangiopericytoma of the small bowel should include segmental small-bowel resection, diagnosis by frozen-section examination of the tumour at the time of operation, a search for metastases and long-term follow-up.

We wish to thank Dr. C. S. Houston, Department of Radiology, University Hospital, Saskatoon, for his interpretation of the radiographs and Dr. G. W. Cates and Dean D. F. Moore, Department of Pathology, for assistance with the pathological diagnosis.

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Résumé

Le rapport décrit un hémangiopéricytome de l'iléum terminal accompagné d'une invagination iléocolique qui est survenu chez une femme blanche de 72 ans. L'invagination a été réduite et la lésion excisée par résection de la portion malade du grêle. La malade était encore bien portante deux ans après l'opération.

Une recherche approfondie de la littérature pertinente ne nous a permis de découvrir que quatre autres cas d'hémangiopéricytome du grêle. Une de ces tumeurs touchait le jéjunum et était également accompagnée d'invagination. Ces malades n'ont pas été suivis comme ils l'auraient dû, mais on sait qu'un malade est mort de métastases hépatiques 18 mois après l'opération et qu'un autre en était à la phase terminale huit ans après l'opération initiale.

Il est clair qu'il faut déceler précocement cette lésion et la traiter convenablement. Dans le traitement préconisé pour l'hémangiopéricytome du grêle, on conseille une résection d'un segment du grêle, un diagnostic basé sur l'examen histologique par congélation et la recherche systématique des métastases et une longue post-observation.

ENTEROLITHS IN MECKEL'S DIVERTICULUM: REPORT OF A CASE AND REVIEW OF THE LITERATURE*

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THE incidence of Meckel's diverticulum in the general population is about 2%. Some complications resulting from this congenital anomaly are well recognized; of the rarer ones, the formation of calculi (enteroliths) in the diverticulum is the least common. Boldero[1] collected only 16 cases from a comprehensive review of world literature, and added one of his own. Since that report, only six more cases have been recorded. If two cases[2, 3] overlooked by Boldero are included the total number of reported cases is 25 (Table I).

TABLE I.—PREVIOUSLY PUBLISHED REPORTS OF ENTEROLITHS IN MECKEL'S DIVERTICULUM

Case	*Authors*	*Age (years)*	*Sex*	*Provisional diagnosis*	*Operative or autopsy findings*	*Remarks*
1	Beal (1852)[4]	14	M	Peritonitis	Autopsy finding of 2 calcified concretions in diverticulum	Cherry stone and orange pips also present
2	Beach (1896)[9]	62	F	Pelvic fistula (vesico-intestinal)	Solitary stone in diverticulum	—
3	Galeazzi (1898)[10]			Original article not consulted		
4	Hollander (1906)[11]	—	M	Perforated appendix	Multiple stones causing perforation of diverticulum	—
5	Znojemisky (1907)[12]	18	M	Perforated appendix	Solitary stone in diverticulum	Appearance similiar to gallstone
6	Sherrin (1909)[13]	38	M	Appendicitis	Narrow-neck diverticulum filled with dark concretions	—
7	Carless (1909)[14]	23	M	Perforated appendix	Solitary stone in diverticulum	—
8	Drummond (1913)[15]	27	M	Acute intestinal obstruction	Single dark-brown calculus	—
9	Quénu (1921)[5]	48	M	Incarcerated right inguinal hernia	Diverticulum in hernial sac contained oval stone	Stone had a central nidus of plum stone. Note resemblance to present case
10	Hanke (1933)[6] and Gerlach (1934)[7]	69	M	Congestive heart failure	Autopsy showed 15 stones in diverticulum but none in gallbladder	Gerlach proved by analysis that these stones could not originate in gallbladder
11	Grevillius (1940)[16]	33	M	Acute abdomen	5 large calculi	Analysis showed calcium carbonate with traces of bilirubin and cholesterol
12	Mulsow (1943)[17]	48	M	Acute abdomen	Gangrenous Meckel's diverticulum containing large stones	Visible on radiographs

The following is a report of a 68-year-old married woman in whom a Meckel's diverticulum containing calculi was discovered during operation for elective repair of an umbilical hernia.

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TABLE I (*continued*)

Case	Authors	Age (years)	Sex	Provisional diagnosis	Operative or autopsy findings	Remarks
13	Gile and MacCarty (1943)[18]	34	M	Intestinal obstruction	2 large calcified fecaliths in diverticulum	—
14	Allen and Donaldson (1945)[19]	58	M	Carcinoma of cecum	Many hard bile-stained stones in large and small sizes	History of cholecystectomy 12 years earlier
15	Basile and Elfersy (1951)[2]	49	M	Carcinoma of sigmoid colon	Single stratified calculus with all gross characteristics of gallstone	Cholecystectomy for biliary disease at age 31
16	Appell and Klotz (1952)[20]	32	M	Acute appendicitis	Numerous hard brown stones	—
17	Appell and Klotz (1952)[20]	39	M	Acute appendicitis	Multiple concretions less than 1.4 cm. diameter	—
18	Duncan (1956)[3]	43	M	Acute appendicitis	Mesenteric abscess due to perforation of diverticulum. Solitary enterolith found in abscess	—
19	Boldero (1958)[1]	61	M	Chronic cholecystitis	Large number of brown faceted calculi	Valve-like fold of mucosa at neck of diverticulum. Gallbladder contained small stones
20	Bergland, Gump and Price (1963)[21]	73	F	Diverticulitis	Solitary enterolith obstructing terminal ileum	Meckel's diverticulum chronically inflamed containing flakes of same material as enterolith
21	Frostberg and Vang (1963)[22]			Original article not consulted		
22	Schlögelhofer (1964)[23]			Original article not consulted		
23	Williams (1965)[24]	81	M	Gastric ulcer	Incidental finding of Meckel's diverticulum containing multiple greenish faceted stones during elective Billroth I gastrectomy	Analysis of enterolith showed 95% organic matter and 5% inorganic, mostly calcium
24	Caridis and Smith (1965)[25]	—	—	Small-bowel obstruction	Original article not consulted	
25	Feldman (1966)[26]	47	M	Chronic cholecystitis	Analysis showed gallstone-like calculi containing calcium phosphate and cholesterol	Stones visible on radiological examination
	Sharma and Benson (present case)	68	F	Umbilical hernia	Diverticulum in hernial sac contained multiple stones	Gallbladder free of stones

Case Report

The patient was first examined on December 7, 1966. She complained of periodic attacks of abdominal discomfort, flatulence, nausea and occasional vomiting over the past three years. A previous gastrointestinal series was reported as normal. She was an obese woman with a moderate-sized umbilical hernia.

After her weight had been reduced by 20 to 25 lbs. she was admitted to the Toronto East General and Orthopaedic Hospital on May 10, 1967, for an elective repair. Results of the routine laboratory investigation were within normal limits. At operation on the

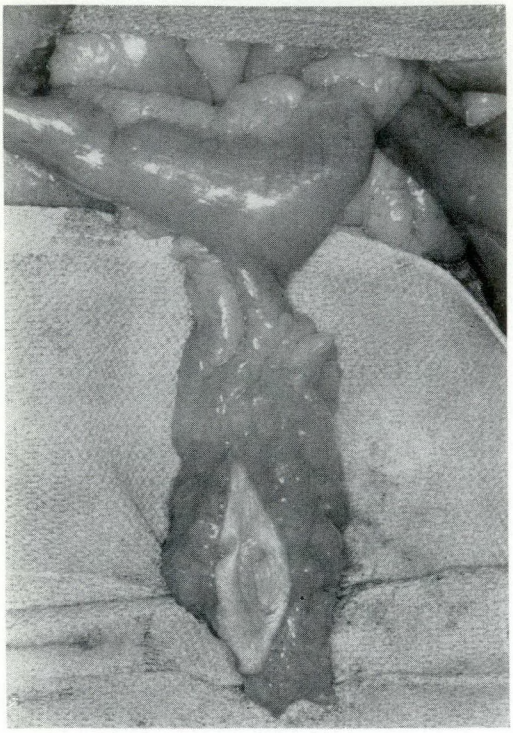

Fig. 1.—Meckel's diverticulum in the sac of an umbilical hernia.

following day she was found to have a long Meckel's diverticulum which was partially contained within and firmly attached to the sac of the umbilical hernia (Fig. 1); it contained six black faceted stones (Fig. 2). The diverticulum was excised and the hernia repaired. Care was taken to inspect the gallbladder which was normal in appearance and contained no stones.

The patient's recovery was uneventful and she was discharged from hospital free from symptoms on the twelfth postoperative day.

Discussion

Only 25 cases of enteroliths in Meckel's diverticulum have been been recorded in the literature since the first case was described by Beal[4] over 100 years ago. This is evidence of the rarity of this complication of Meckel's diverticulum, which itself is found in only 1% to 2% of the general population. All previously recorded cases, with the exception of two, were in men, indicating a marked difference in the sex incidence. This is only the third case in a female patient.

Another unusual feature was the pres-

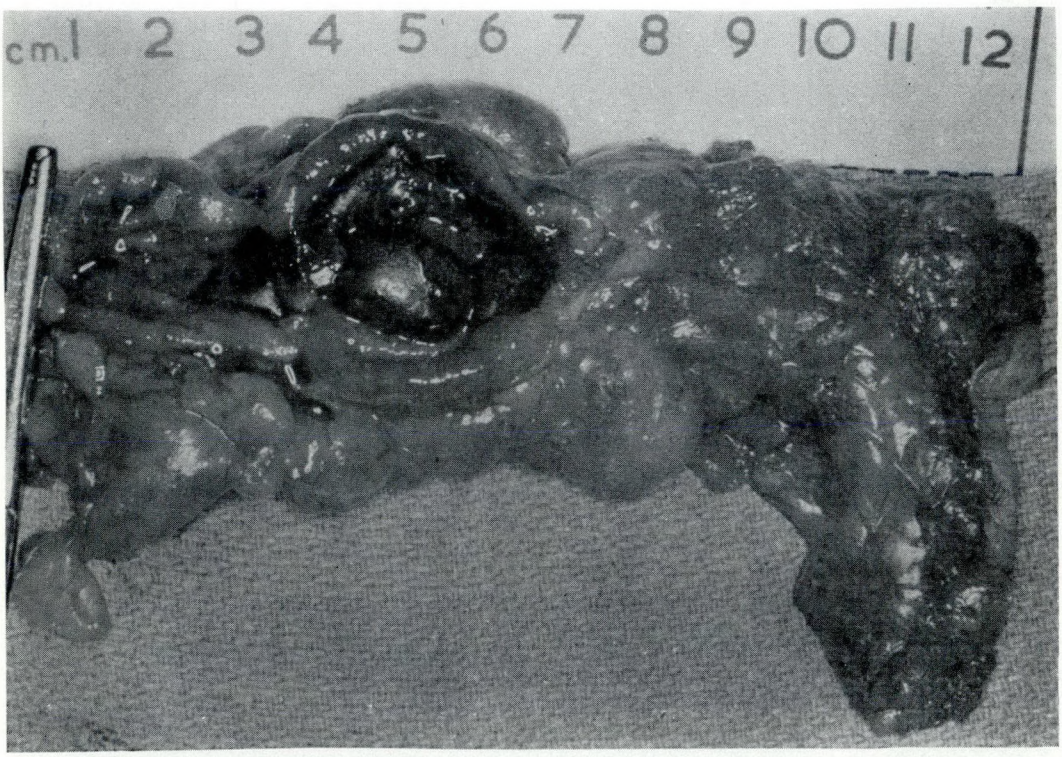

Fig. 2.—Diverticulum opened after excision. Multiple black faceted stones are seen. On palpation at laparotomy the gallbladder seemed to be normal.

ence of the diverticulum in a hernial sac. In this it bears some similarity to the case described by Quénu[5] in which the diverticulum was found in the contents of an irreducible right inguinal hernia.

The stones in this case closely resembled gallstones: they were hard, multiple, faceted and black. The gallbladder itself was free of stones and was not grossly diseased. This was the case in most of the previous accounts of enteroliths, except Hanke's[6] where calculi were found in the gallbladder as well as in the diverticulum. Unfortunately an analysis of the stones from our patient was not done. Gerlach[7] and others have found wide variations in the composition of enteroliths. In most this is indistinguishable from that of gallstones. Others report the presence of zinc, lead, copper, iron, manganese or silica.

The etiology and pathogenesis of lithiasis in Meckel's diverticulum is intriguing. Most diverticula are of the wide-neck variety and contain smooth muscle capable of peristalsis in their wall, so that there is little chance of stagnation of intestinal contents. Should the neck of the diverticulum be narrow, if a flap of mucosa provides a valve-like mechanism at the neck, or if inflammation (diverticulitis) is present, any of these circumstances may contribute to stasis of intestinal contents in the diverticulum. The presence of a foreign body like a tomato skin, plum stone or small gallstone (which may have escaped from the biliary tree) may provide a nidus for the formation of an enterolith within a Meckel's diverticulum. All of these are important factors which may well be related to the pathogenesis of this extremely interesting but rare complication.[8]

Summary

A 68-year-old woman with a Meckel's diverticulum containing enteroliths is described, and the pertinent literature is reviewed.

We wish to thank Dr. C. T. Robertson, Surgeon-in-Chief, Toronto East General and Orthopaedic Hospital, for permission to publish this case.

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Résumé

Présentation d'un cas d'entérolithes dans le diverticule de Meckel, calculs découverts chez une femme de 68 ans. Nous n'avons pu retrouver que 25 cas semblables (dont deux seulement chez la femme) dans la littérature chirurgicale.

Cette femme avait été vue pour la première fois à la consultation chirurgicale pour une hernie ombilicale. Elle se plaignait depuis trois ans de malaises abdominaux, de flatulence, de nausée et parfois de vomissement. On constata, à l'examen, qu'il s'agissait d'une obèse dont la hernie ombilicale était de dimension moyenne. Six mois plus tard, qu'elle avait perdu un poids considérable, on procéda à la réparation élective de la hernie. Au cours de l'opération, on découvrit un long diverticule de Meckel, contenant six calculs noirs à facettes multiples. Le diverticule était partiellement contenu et fermement fixé au sac herniaire. On procéda à son excision et la malade reçut son congé au douzième jour post-opératoire après une convalescence sans incident. Jusqu'à présent, elle est restée asymptomatique.

Les auteurs abordent sommairement l'étiologie de la formation de ces entérolithes et donnent un résumé des caractéristiques principales des cas déjà rapportés. Il semble que l'infection, la stase et la présence d'un corps étranger servant de noyau soient des facteurs importants dans la pathogénie de l'entérolithiase, tout comme ils le sont dans la formation de calculs ailleurs dans l'organisme.

TOTAL AGANGLIONOSIS OF COLON

Total aganglionosis of the colon is uncommon, but rather than being extremely rare, it is often unrecognized. Before 1967, 87 patients with typical aganglionic disease of the distal colon and 13 patients with Hirschsprung's disease of the entire colon were seen at a children's hospital. The ages of the 13 children at the time of diagnosis ranged from 5 days to 12 months, with a median age of 23 days. Nine of these children were male, and four were female. None had other congenital defects of ectodermal origin. One child died while being prepared for operation, but 7 of the 12 who were operated upon recovered.

Early symptoms and signs of total aganglionosis of the colon include delayed and persistent meconium stools, progressive vomiting and abdominal distension, failure to gain weight, evidence of abdominal distress, elevated leukocyte counts, and so-called obstructive diarrheal stools. Later all of these patients became dehydrated and debilitated, and at this time, bowel sounds become hypoactive in most patients. Visible peristalsis and palpable intestinal loops were observed in 6 of these 13 patients; three patients had dilated colons. Roentgenographic examination revealed dilated loops of small intestine in all patients, and contrast studies of the colon revealed that the lumen was normal in seven patients and dilated in two. Partial ileal obstruction associated with stasis of the colon is almost diagnostic of aganglionosis of the colon in the newborn. Rectal biopsy confirms the diagnosis, but does not show its extent. Lesions that may simulate total aganglionic disease of the colon include "short-segment" disease, pancreatic fibrosis with impaction of meconium, malrotation of the colon with volvulus, and the fetal distress syndrome associated with local or general hypoxia.

In this series every patient treated by prolonged conservative measures has died. Deficiencies of blood, fluid and electrolytes should be corrected before operation. The objective of the first operation should be to relieve the obstruction and, if the situation permits, to establish the lowest level to which normal intestine extends. A decompression ileostomy is preferable; ileotransverse colostomy or cecostomy will not relieve the obstruction. Antibiotics should be given, particularly when there is contamination. Addition of 0.5 g. sodium chloride and 0.5 g. sodium bicarbonate to each 24 oz. of formula will compensate for ordinary losses of electrolytes from the ileostomy. At the second operation an ileoproctostomy is performed from an abdominoperineal approach. Later the bypassed aganglionic colon and terminal ileum may be removed electively.—Soltero-Harrington, L. R., Garcia-Rinaldi, R. and Able, L. W.: Total aganglionosis of the colon; recognition and management, *J. Pediat. Surg.,* **4:** 330, 1969.

MANAGEMENT OF INGESTED FOREIGN BODIES*

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A FOREIGN body in the gastrointestinal tract can almost always be managed conservatively.[1] Operation is necessary only if any complications arise while the patient is under observation in hospital. Sharp-pointed metallic foreign bodies are less likely to cause difficulties than are fish or meat bones.[2]

A case is described in which a patient swallowed a large number of open safety pins and passed them per rectum without the development of any complications.

CASE REPORT

A 13-year-old white girl was first seen in the emergency department on September 7, 1968, when it was declared that she had swallowed a total of 17 safety pins on three different occasions in the four days before admission. The pins had been opened, straightened and swallowed with the blunt ends first. The length of the opened-out pins was approximately 2¼ inches.

The patient had been undergoing psychiatric treatment and had previously attempted to commit suicide. She was taking phenobarbitone to control grand mal epileptic seizures. She had had an abortion three months before admission.

When interviewed the patient complained of colicky abdominal pains which were felt chiefly in the umbilical region. There was generalized abdominal tenderness on gentle palpation, but the abdomen was soft, without guarding or rigidity. On auscultation bowel sounds were normal. The open safety pins were clearly evident on radiographic examination; about half of them lay in the region of the stomach, with the remainder scattered through the abdominal cavity (Fig. 1). There was no free gas under the diaphragm (Fig. 1). She was admitted to hospital, given a full diet and kept under close observation.

She complained of colicky abdominal pain for the next two days and on the third day she had a stool which contained one metal ring, two bobby pins, five open safety pins and large quantities of thread. Films taken at this time, when compared to those taken at the time of admission, showed a change in the position of all the pins (Fig. 2). She passed

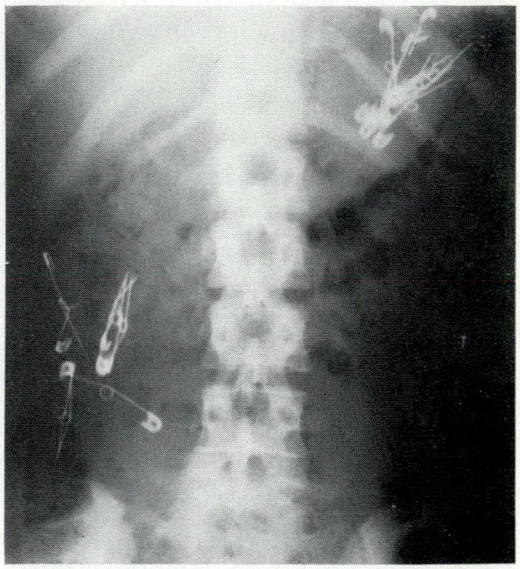

Fig. 1.—Radiograph taken on admission to the emergency department showing the distribution of the safety pins. There is no free air under the diaphragm.

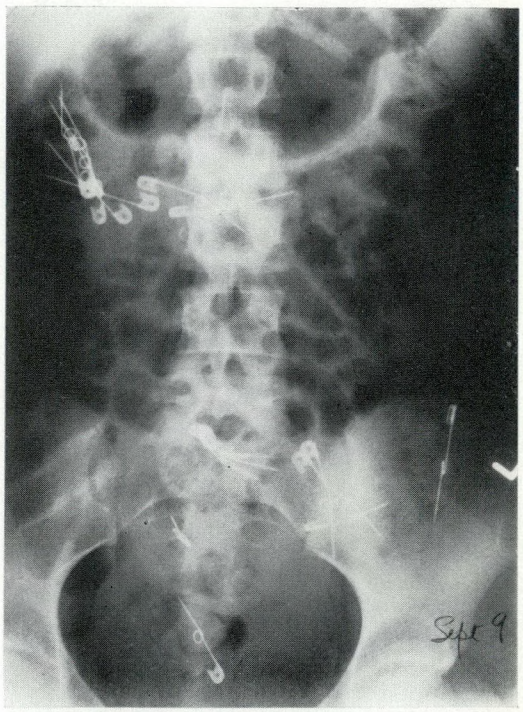

Fig. 2.—On the third day after admission the position of the pins has changed as they pass through the gastrointestinal tract.

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the remaining pins on the next two days, but radiographs taken three days later (September 15) showed an open paper clip lying in the region of the stomach (Fig. 3). This was passed per rectum on the following day.

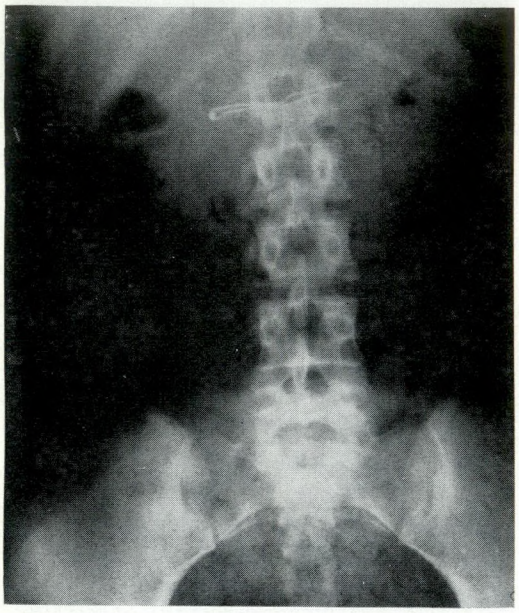

Fig. 3.—On September 15 only one open paper clip remained in the gastrointestinal tract. All the safety pins had been passed per rectum three days before.

Discussion

Most swallowed foreign bodies that reach the stomach are passed uneventfully per rectum. The sites at which their passage may be impeded are: (1) the physiological sphincters—cardiac, pyloric and ileocecal—and (2) those sites where the foreign bodies must negotiate a curve in the gastrointestinal canal, e.g. the duodenal curve, the duodenojejunal junction, the rectosigmoid junction or where the bowel occupies a hernial sac. Congenital anomalies and bands may also obstruct the passage of a foreign body. Finally, foreign bodies can lodge in an appendix or a Meckel's diverticulum and give rise to the clinical picture of acute appendicitis.[3]

Gross[4] estimated that approximately 7% of patients who had swallowed foreign bodies required an operation. According to Stevenson and Hastings,[5] 75% of ingested foreign bodies reach the stomach and of these, 90% pass successfully through the gastrointestinal tract. They reviewed 113 cases in infants and children of whom 26 needed laparotomy. Operation was performed most often to retrieve safety pins or hair pins; in only two cases had the bowel been perforated. Open safety pins are usually swallowed blunt end first so that almost all of them reach the stomach. In most cases laparotomy was indicated because radiographs showed that progress of the foreign body had become arrested.

A metallic foreign body such as an open safety pin is less likely to give rise to trouble because, unlike swallowed fish or meat bones, there is a definite history of its ingestion, it is easily seen on radiographs and its progress can be followed.[2]

The complications that may arise from the ingestion of a foreign body are either perforation of the bowel or hemorrhage. Perforation rarely gives rise to general peritonitis because the process is a gradual one; inflammation develops and there is time for abscess formation. There may be perforation of the appendix or a Meckel's diverticulum. Perforation of the bowel within a hernial sac is possible. Any hemorrhage that occurs is rarely of a major degree.[6]

In the treatment of patients who have swallowed foreign bodies we recommend: (1) that daily radiographs of the abdomen be taken to assess the progress of the body through the gastrointestinal tract; (2) that a full high-residue diet be given (so that the foreign body will be embedded in the bulky stools); and (3) that a close watch be kept for clinical signs of perforation of, or hemorrhage from, the gastrointestinal tract.

The indications for operation are: (1) failure of progress of the foreign body as shown on repeated x-ray films; (2) perforation of the bowel; (3) hemorrhage from the gastrointestinal tract; and (4) known congenital anomalies of the gastrointestinal tract.

Summary

An unusual case of ingested foreign bodies is presented. The mode of presentation, complications and treatment of foreign bodies in the gastrointestinal tract have been outlined.

We are most grateful to Dr. D. Adamson of Edmonton, for permission to publish this case report.

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RÉSUMÉ

Les auteurs présentent un cas exceptionnel d'ingestion de corps étrangers et exposent le mode de présentation de ceux-ci dans le tractus gastro-intestinal, les complications possibles et le traitement.

HEPATIC LOBECTOMY IN CHILDHOOD

Although the benefits of irradiation and chemotherapy in the treatment of certain neoplasms of childhood have been well documented, these modalities are not without toxic effects. The authors present clinical and experimental evidence which suggests that the toxicity of combined roentgen ray and actinomycin D therapy is exaggerated after hepatectomy. In the past five years, two children underwent hepatic lobectomy for localized liver metastases following nephrectomy for Wilms' tumour at one hospital.

The first was a 5½-year-old boy who had a nephrectomy for Wilms' tumour which had metastasized to the lung, renal vein, and regional lymph nodes. Postoperative irradiation and actinomycin D were given. Six months later a right hepatic lobectomy was performed because of a solitary liver metastasis, and actinomycin D and irradiation were again given. Postoperative complications included intra-abdominal bleeding associated with thrombocytopenia, recurrent collections of blood, bile, and pus in the right subphrenic space and, later, liver dysfunction. Recurrent lung metastases were treated with actinomycin D and radiation and a severe skin reaction developed. Over the subsequent four years progressive severe impairment of normal growth and development associated with persistent abnormal hepatic function has developed. When ascites developed during the past year, an open liver biopsy was performed which demonstrated hepatic fibrosis but no tumour.

The second patient, a 3-year-old girl, underwent nephrectomy for Wilms' tumour. No metastases were seen at operation. Actinomycin D and irradiation were given postoperatively. Three years later a right hepatic lobectomy was performed because of a solitary metastasis, and actinomycin and irradiation were given. Postoperatively, a severe cutaneous radiation reaction was noted and diarrhea, severe leukopenia, and thrombocytopenia developed. In the next few months ascites appeared. Liver biopsy showed increased fibrosis in the portal and central areas and abnormal lobular patterns which were compatible with late effects of radiation therapy. The child now has a non-resectable intraperitoneal tumour.

In both children complications occurred after the use of routine and usually safe doses of irradiation and actinomycin D. Liver function was decreased in both when these agents were given.

Experimental studies were carried out on 97 adult male Sprague-Dawley rats who had undergone 70% hepatectomy. One group received no further treatment, one group had irradiation to the liver, the third group received actinomycin D, and the fourth group had irradiation to the liver and actinomycin D. Four similar groups of rats who did not undergo hepatectomy served as controls. The overwhelming majority of deaths occurred in the animals who had hepatectomy and were treated with actinomycin D. The addition of irradiation did not appreciably increase the high mortality rate of 70%. The only animals in the non-surgical groups which died were those which had been treated with both actinomycin D and irradiation. Death rates, body weight changes, and histologic studies confirm the impression that partial hepatic resection decreases tolerance to this drug and irradiation, and that these modalities in turn limit liver regeneration.

The authors suggest that in children undergoing partial hepatectomy, chemotherapy and irradiation be withheld until liver regeneration is nearly complete.—Filler, R. M. *et al.*: Hepatic lobectomy in childhood; effects of x-ray and chemotherapy, *J. Pediat. Surg.*, **4:** 31, 1969.

IDIOPATHIC CYSTIC DILATATION OF THE COMMON BILE DUCT: REPORT OF A CASE IN AN ADULT*

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CONGENITAL cystic dilatation of the common bile duct is a rare condition. Until about 10 years ago only 403 authentic cases had been reported in the world literature.[1] To illustrate the rarity of this condition Smith[2] was able to find only two cases in 757,000 admissions to the Presbyterian Hospital, New York. Most of the reported cases have been in children. The condition is extremely uncommon in adults and from a study of these case reports[1-3] it is often impossible to determine whether the condition was congenital or acquired. The following report describes our experience with a man who had idiopathic cystic dilatation of the common bile duct, which posed some diagnostic difficulty. We have used the term "idiopathic" rather than "congenital" dilatation because in our patient there was no evidence of a developmental defect.

CASE REPORT

J.M., a 70-year-old man, had been admitted to hospital four years before because of cirrhosis of the liver and upper gastrointestinal bleeding, presumably from esophageal varices. At that time an upper gastrointestinal barium study did not reveal any abnormality in the duodenum or any filling defects.

His most recent admission, on October 2, 1967, was for mild generalized abdominal pain of three days' duration, which was not related to food intake. He had no other signs or symptoms and denied having ever been jaundiced.

On physical examination there was slight obesity, but no other obvious abnormality. His blood pressure was 150/90 mm. Hg, his hemoglobin was 13.6 g./100 ml. Routine laboratory examination was non-contributory except that his stool was weakly positive for occult blood. The total serum bilirubin was 0.9 mg./100 ml. and serum amylase was 185 Somogyi units/100 ml. Prothrombin time, serum proteins and alkaline phosphatase were all normal. Liver scan showed an enlarged liver. An upper gastrointestinal series showed two filling defects in the duodenum, one in the first and the other in the third part (Fig. 1). When this investigation was repeated two days later only the defect in the third part was evident; the radiologist suggested that the lesion might be extramucosal (Fig. 2). Oral cholecystograms done on two occasions showed a non-functioning gallbladder. Our preoperative diagnosis was chronic cholecystitis with lithiasis and lipomas of the duodenum.

Fig. 1.—Upper gastrointestinal series showing filling defects in the first and third portions of the duodenum.

At operation the gallbladder was found to be markedly fibrotic and to contain numerous multifaceted stones. There was a fistula between the gallbladder and the duodenum. A cholecystectomy was performed. No cholangiogram was done because we considered it was unnecessary in the absence of a history of jaundice, and because of the small calibre of the cystic duct, and the relatively large stones.

When we opened the anterior wall of the third part of the duodenum, we found an intramural mass. The mucosa of the posterior

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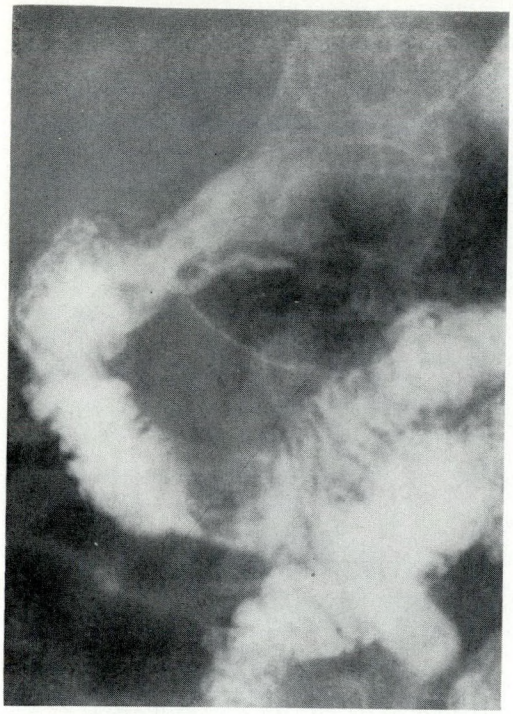

Fig. 2.—Upper gastrointestinal series showing filling defect in the third portion of the duodenum.

wall was then incised and thin bile poured out. The cavity beyond, i.e. the dilated common bile duct, was more than 3 cm. in diameter. The dilatation extended up to about the point of entry of the cystic duct; beyond this the diameter of the common duct diminished to normal. The duct wall was 3 to 4 mm. thick. The incision was enlarged and the cyst wall was sutured to the duodenal wall to allow drainage. The resulting stoma was about 2 cm. long. Biopsy of the cyst wall showed chronic inflammation and fibrosis. The liver was grossly cirrhotic and microscopic examination identified fatty change with chronic inflammation and fibrosis. The picture was consistent with biliary fibrosis secondary to obstruction of the bile ducts.

The patient's postoperative course was uneventful. When he was seen a year later in the outpatient clinic he was in good health.

Discussion

This patient had a congenital or idiopathic cystic dilatation of the common bile duct. In the classification of Alonso-Lej, Rever and Pessagno,[1] this lesion belongs to the first type of congenital cystic dilatation of the common bile duct. (The second type is congenital diverticulum and the third type is congenital choledochocele. All three are grouped as "congenital choledochal cysts".)

This patient's cyst was relatively small; usually such cysts contain one to two litres of bile. The largest recorded in the literature was that reported by Browne.[4] After 10,000 ml. had been removed by three abdominal "taps", the cyst still contained 13,340 ml. at the time of operation.

We had difficulty in making the correct diagnosis in the present case because two filling defects appeared in the radiograph of the duodenum; usually there is only one, in the second portion. After looking at the artist's conception (Fig. 3), it is easy to visualize how the defects were produced. One clue to the diagnosis was the disappearance of the defect in the first portion of the duodenum two days after the first radiological examination. Presumably, when the radiographs had been taken four years previously the cyst was empty, so that no filling defects were visualized.

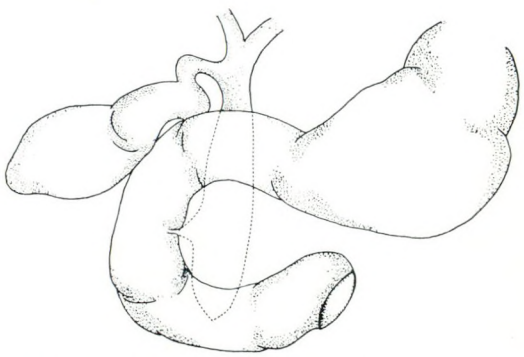

Fig. 3.—Artist's reconstruction of the pathologic picture to explain the filling defects in the duodenum in the upper gastrointestinal series.

The possibility of a pseudocyst of the head of the pancreas was entertained, but the presence of bile in the cystic cavity made this unlikely.

The etiology of this group of anomalies is still debated. Some surgeons believe that they are congenital, resulting from a weakness in the wall. Others contend that they arise owing to acquired obstruction to the outflow of bile at the choledochoduodenal junction. Gross[5] believes that this is a lesion of multiple etiology and that a single concept cannot explain all cases. We have

chosen, therefore, to use the term "idiopathic" in the present case.

The condition occurs most frequently in women under the age of 25 years. The clinical picture varies widely. Many patients are asymptomatic. Some cysts compress surrounding structures or may rupture causing peritonitis. The classical triad of idiopathic cystic dilatation of the common bile duct is said to comprise abdominal pain, a right upper quadrant mass and jaundice.

With respect to treatment no agreement has been reached. Some surgeons prefer sphincterotomy; others, excision of the redundant cyst wall and repair; still others perform some form of bypass from the gallbladder or common bile duct to some part of the intestinal tract (stomach, duodenum or jejunum). Because most reports are of individual cases, and because the methods of dealing with the condition are varied, the mortality rate also varies. However, the overall mortality rate is approximately 10%.[1] Complications include rupture, causing peritonitis, and obstructive biliary cirrhosis.

Summary

A case of idiopathic cystic dilatation of the common bile duct in an adult is described. Some difficulty in diagnosis was encountered. A cystduodenostomy was performed. The patient is well a year after operation. The literature concerning this condition is briefly reviewed.

We gratefully acknowledge the help of Dr. J. Halls of the Department of Radiology.

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Résumé

Les auteurs présentent un rare cas de dilatation kystique idiopathique du cholédoque.

Il n'avait pas été possible de diagnostiquer la pathologie avant d'opérer, en raison des difficultés d'interprétation des radiographies. Les repas barytés des voies digestives supérieures avaient mis en évidence deux vices de remplissage dans les première et troisième portions du duodénum.

On a procédé au drainage de la cavité kystique dans le duodénum au moyen d'une kyste-duodénostomie. Un an après l'opération, le malade n'avait aucun symptôme.

L'article se termine par une brève revue de la littérature sur cette pathologie.

PYOGENIC LIVER ABSCESS

Liver abscesses in 48 patients were seen at two British hospitals in a 10-year period. In 17 abscesses were related to another acute illness, ran a short course, were often multiple, and were diagnosed and treated in a fashion similar to other postoperative intra-abdominal pus collections.

In 31 patients a more chronic presentation was noted, the abscesses were usually solitary and the primary source of infection was often obscure. Weight loss and anemia were seen in all patients in this group; hepatomegaly was seen in 73%, fever and leukocytosis in 66.6%, pain in 57%, chills in 40% and jaundice in 23%.

A dropping serum albumin level and a grossly elevated serum vitamin B_{12} level were the most reliable laboratory tests. A low leukocyte count, coma, and low blood urea nitrogen level were associated with the poorest results. Sixteen patients had *Escherichia coli* cultured from their abscesses, but the authors stress the importance of both aerobic and anaerobic culture. Isotope and ultrasonic scanning of the liver was performed in a few instances and was quite helpful in localizing the lesion or lesions.

Fourteen patients had metastatic abscesses, usually in the lung or brain; only five of these patients survived. Of 22 patients undergoing drainage, eight eventually died. Of nine patients without drainage, all died.

The authors stress the importance of suspicion of this lesion in cryptogenic fever and re-emphasize the importance of early localization and drainage.—Butler, T. J. and McCarthy, C. F.: Pyogenic liver abscess, *Gut*, **10:** 389, 1969.

EXPERIMENTAL SURGERY

AGE CHANGES IN LUMBAR INTERVERTEBRAL DISCS*

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Vancouver, B.C.

In 1932 Schmorl and Junghanns[1] published their classic monograph "The Human Spine in Health and Disease"; little has been added since then regarding normal disc morphology.

The purpose of this paper is to review, chronologically, the life history of various components of the disc. The normal, "predegenerate", anatomy will be defined and the degenerative patterns will be analyzed in an unselected series of cases. An attempt will be made to correlate relevant data and to fill in certain gaps in knowledge of the subject through reference to our own study. We believe that the picture of pathogenesis presented gives a strong implication of its cause.

Material and Methods

From the autopsy room at the Vancouver General Hospital, 59 subjects were selected at random and were separated into 10-year age groups for the purposes of discussion. The age at death ranged from birth to 80 years (Fig. 1).

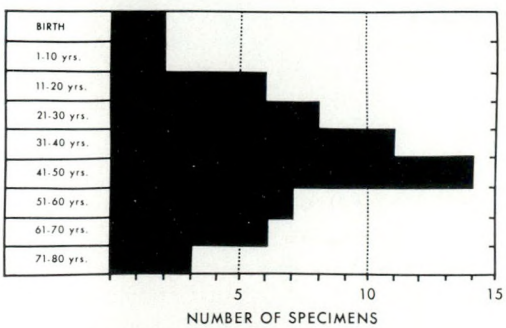

Fig. 1.—Age distribution of cases.

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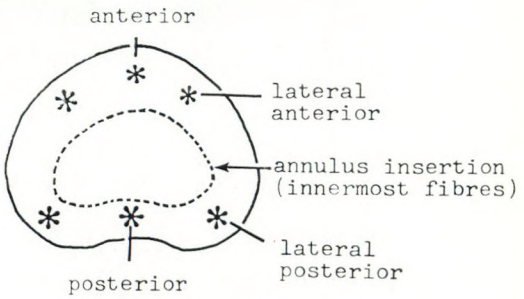

Fig. 2.—Annulus test areas.

The lumbar vertebrae were removed "en bloc" and about one-third of these were discographed using an anterior approach. The specimens were then refrigerated at 5° C. and dissected several days later.

The specimens provided 296 individual discs. Initially they were cut transversely, then dissected completely using a blunt and sharp technique. Some specimens were dissected from the sagittal plane.

Comparative softening of various areas in the annulus (Fig. 2) was measured with a Shore durometer (hardness type A-2 ASTM D760) (Fig. 3), adapted to measure the type of resistance encountered.

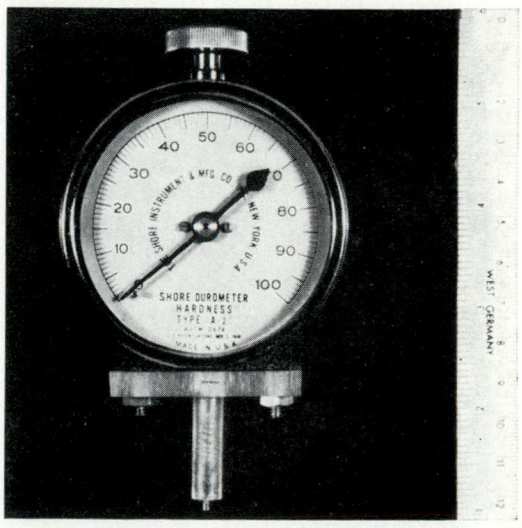

Fig. 3.—Shore durometer.

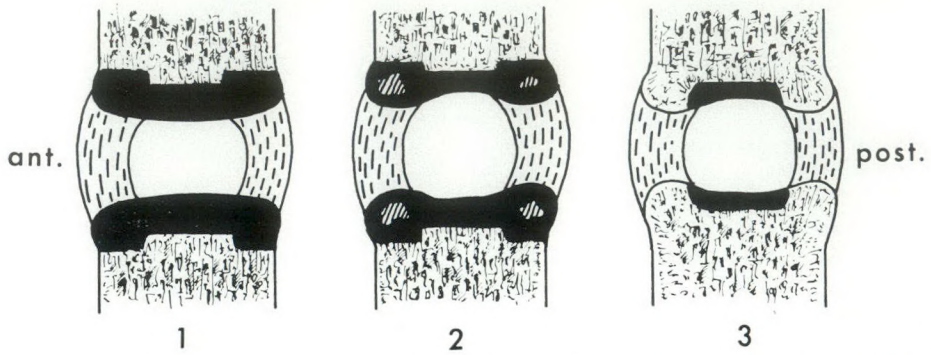

Fig. 4.—Developing annulus and secondary epiphysis. Vascular connections from the nucleus to the vertebra are not indicated.

Experimental Anatomy

In the spinal anlage certain cells in the intervertebral region differentiate first into chondroblasts, then into fibroblasts, forming fibrocartilaginous bands or lamellae. They are attached to the longitudinal ligaments peripherally and to the cartilage of the vertebral bodies above and below. These lamellae (annulus fibrosus) are attached to an area of cartilage destined to become the secondary epiphysis, or epiphyseal ring, and take little if any attachment from the area of cartilage that will become the central cartilage plate (Fig. 4).

The surface of the cartilaginous body at birth is convex in all directions, but as the secondary ring epiphysis develops the surface takes on the more concave appearance characteristic of the adult, providing less mobility and more stability. The ring epiphysis is more heavily developed anteriorly than posteriorly.[2]

Originally notochordal cells are in continuity and have a vascular connection with the primordia of the vertebral body through the central cartilage plate area. As the cartilaginous vertebral body develops these vascular connections are obliterated, but evidence of their past remains as well-marked, concentrically arranged "pits" on the cartilage plate of the body. These are covered by a thin layer of cartilage at birth. At this time the nucleus is a gel and the anterior and posterior lamellae are of the same height and thickness; the disc is very thick compared to the bodies and the spine is in a slightly flexed attitude.

As the end of the first decade approaches, certain changes become evident. The annulus is of greater vertical thickness anteriorly than posteriorly and the posterior lamellae are arched posteriorly and compressed by their narrowed bony confines. However, as at birth, the actual number of anterior and posterior lamellae remains the same (shown by actual count in our present series).

The nucleus at 10 years appears as a gel with a few scattered fine granulations. Lying directly on spongiosa, the thick cartilage plate of the vertebra appears undisturbed, its pits still obliterated. From this point, however, changes occur leading towards degeneration.

Degenerative Changes

At a given age changes differ with the disc level. For the purpose of this study, we shall consider the typical findings at L4-5 with overall changes slightly advanced at L5-S1, and of slightly less degree at levels higher than L4-5.

10 to 20 Years of Age

Vascular tissue begins to invade the nucleus early in the second decade. The thin cartilage overlying the concentric pits is destroyed by vascular proliferations from the vertebral body (Fig. 5). Fine strands of connective tissue course through the nucleus. However, it remains functionally as a mobile gel, bulging when cut across and retaining a clearly demarcated though somewhat less abrupt junction with the surrounding annulus.

As a lordotic curve develops, the posterior lamellae lose height by arching back-

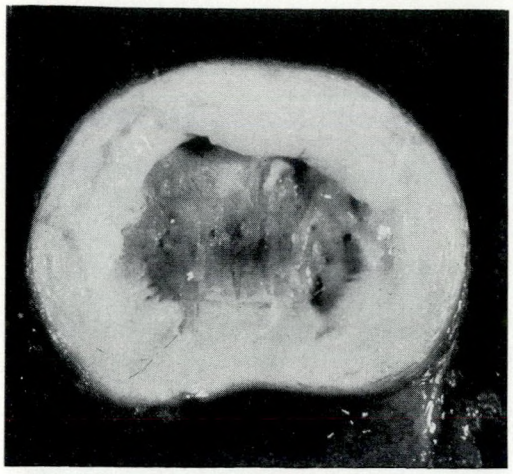

Fig. 5.—11 years. Vascular proliferations through central cartilage pits.

wards and becoming packed together, giving the false impression that there are fewer lamellae posteriorly than anteriorly and presenting an overall picture of a relative movement of the annulus backwards. By the mid-teens, a few of the innermost anterior lamellar sheets begin to thicken and collapse towards the nucleus (Fig. 6).

21 to 30 Years

In the next decade there is a transition of the nucleus from a mobile gel to a fibrous viscous structure, still retaining its volume. Its mobility, however, is decreased

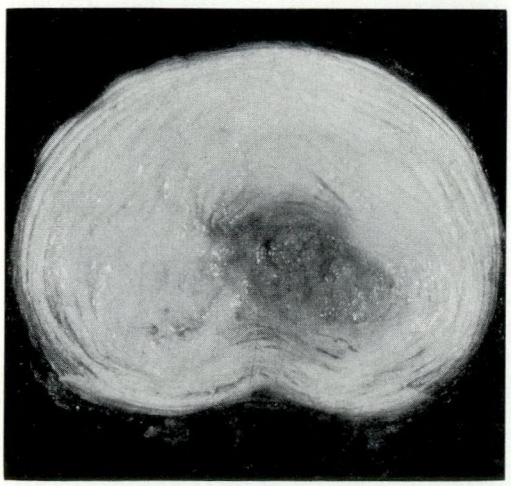

Fig. 6.—11 years. Compression and decrease in height of posterior lamellae give the impression of fewer lamellae posteriorly and relative movement of the annulus backwards. A few innermost anterior lamellar sheets begin to thicken and bulge centripetally.

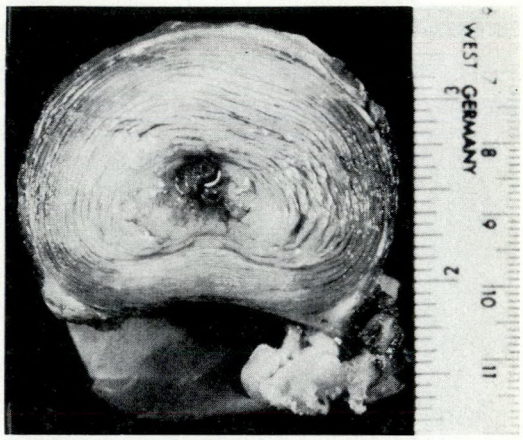

Fig. 7.—24 years. Thickening and collapse of anterior lamellae and compactness of posterior lamellae result in a typical dog-ear corner (toluidine-blue stain).

because of fibrous anchoring to the plate and indistinct fibrous union with the surrounding annulus fibrosus. An area of brownish necrotic degeneration begins to form in the centre of the now fibrous nucleus.

Towards the end of the decade the vascular pits on the cartilage plate enlarge, often filling with the same fibrous tissue that invades the nucleus. This was shown microscopically by Böhmig.[3]

During this time thickening and centripetal bulging of the anterior lamellar fibres continue gradually. The posterior fibres, on the other hand, because of their shortened compact nature, collapse very little and only in a centrifugal direction (Figs. 7 and 8).

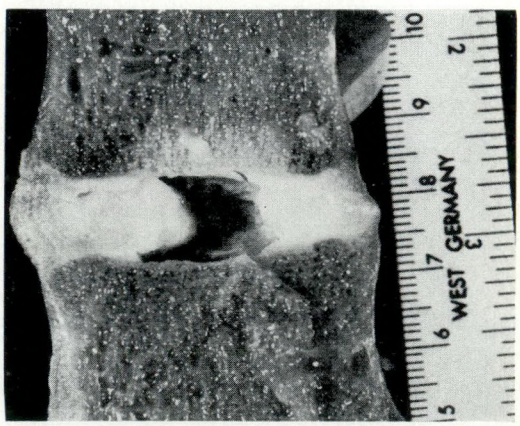

Fig. 8.—24 years, lateral view. Inward bulge of anterior lamellae, outward bulge and compression of posterior lamellae.

The lateral posterior areas of the annulus now constitute junctional zones between the anterior two-thirds of the annulus which is loose and collapsing centrally, and the posterior one-third which is compact and arching centrifugally. The end result is a twisting of the lateral posterior lamellae and a "dog-ear" corner effect, typical of a disc in the third decade.

31 to 40 Years

Small clefts begin to appear between, and running in the direction of, the lamellae. These begin at the mid-height of the annulus and undermine upwards and downwards as far as the lamellar insertions. Such clefting is seen first at the posterior lateral dog-ear corners, and later is evident directly posteriorly.

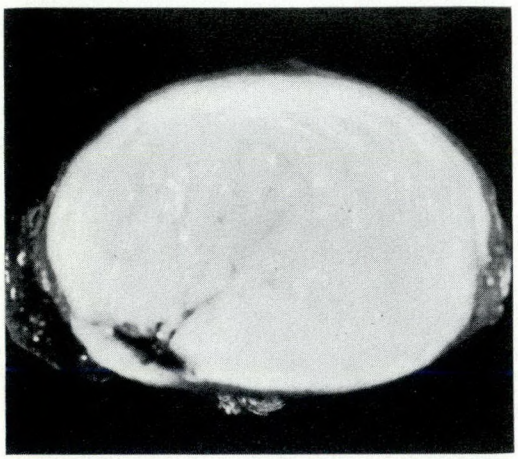

Fig. 9.—32 years, L4-5. Coalescing annular clefts produce a complete radial tear (asymptomatic).

By age 40 granulation pits on the cartilage plate may coalesce producing a larger cavity filled with fibrous or necrotic debris, which is considered an early Schmorl node. Any remaining gel in the nucleus undergoes complete fibrous replacement, but usually retains a good volume, shown by marked bulging when cut across; however, there appears to be a distinct individual variation. The necrotic debris of the nucleus may be a greater or lesser proportion of the total nuclear mass, with a corresponding decrease in volume.

The incidence of annular clefts increases from 30 years onwards, and the clefts frequently coalesce producing a complete radial tear usually at the posterior lateral corners (Fig. 9).

40 to 60 Years

Over the following two decades the cartilage plate as a whole becomes thinned with the development of more granulation defects in the underlying bone. Meanwhile, the nucleus assumes a more pulpy consistence and as apposition of the vertebral bodies progresses, the fibrous parts become more dense and adherent to the surrounding structures (Fig. 10). Annular clefting is virtually always present and begins in the anterior regions of the annulus. Coalescing clefts and complete radial tears at the posterior lateral corners are commonplace and extend to the posterior longitudinal ligament.

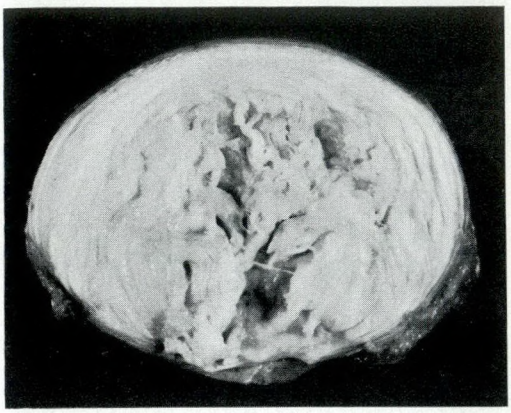

Fig. 10.—48 years. Annular clefting is always present. Coalescing clefts and complete radial tears are common both at the posterior lateral corners and directly posterior as in this case.

The previously compact, tough, though clefted posterior lamellae now begin to break down. This process may result in a large, wedge-shaped defect presenting as a massive radial "tear" extending to the posterior longitudinal ligament. The ligament remains firm at this stage, as it does through all age groups, except for isolated fenestrations and detachments from vertebral borders.

61 to 80 Years

Some remnants of cartilage plate generally persist in advanced age groups. The parts of the nucleus that remain fibrous

TABLE I.—SEM (Standard Error of Mean) for Pressure Readings

Average age (years)	Anterior			Posterior			Lateral anterior			Lateral posterior		
	Average value	SEM	"N"	Average value	SEM	"N"	Average value	SEM	"N"	Average value	SEM	"N"
.6	68.2	3.9	5	60.4	4.1	5	—	—	—	58.8	3.2	5
6.0	47.9	5.9	15	60.0	2.2	15	44.8	2.7	30	45.7	1.9	30
15.6	40.8	3.0	25	45.1	2.1	25	31.7	2.0	50	31.6	1.7	50
27.5	40.3	1.8	20	43.2	3.8	20	33.1	1.4	40	30.0	2.4	40
35.5	35.8	1.9	45	41.7	2.7	45	40.0	2.1	90	31.6	1.5	90
45.0	39.5	1.9	45	37.1	2.3	45	34.3	1.4	90	27.9	1.7	90
53.7	39.9	—	15	24.4	—	15	31.0	2.2	30	19.9	2.2	30
66.0	37.1	—	15	29.5	—	15	32.9	3.8	30	25.8	4.7	30

are difficult to differentiate from true annular lamellae. The latter present an almost totally unorganized picture by 80 years of age when only a few outer lamellae are discernible. The original contoured bony surfaces of the vertebrae gradually flatten and become more or less apposed, separated only by unorganized fibrous tissue. Some cartilage plate remains.

Pressure Studies on the Annulus

While carrying out the dissections in this series it became evident that, whereas gross deterioration and destructive changes were found only in the discs of the over-50 age group, a distinct softening occurred consistently after age 30. This softening was found in the posterior third of the disc where later destructive changes would be added.

In an effort to assess this factor quantitatively, a "hardness" test was carried out on each of the discs studied using a Shore durometer (Fig. 3) specially adapted to measure the type of resistance encountered. In each disc six locations, 5 mm. from the periphery, were tested (Fig. 2). The readings were recorded graphically (Fig. 11).

Each line on the graph represents pressure readings at the following areas: anterior, posterior, right and left lateral anterior (combined), and right and left lateral posterior (combined). The cases are classified in 10-year age groups for which all the pressures at one location are averaged and plotted. Therefore each line represents an average reading for one location, but with no differentiation as to disc level.

Since the anterior readings change the least they are used as a reference point. The compact posterior fibres remain firm until about 45 years of age when they begin to degenerate and soften. Lateral anterior areas most closely parallel the lateral posterior areas and readings remain similar until about 25 years. At this age the readings begin to diverge, since the lateral posterior fibres become weaker.

Table I shows the standard error of the mean value for each point on the graph, where "N" indicates the number of values taken for that point.

The wide bands in Fig. 12 indicate four times the standard error of the mean value at each age group, thus giving a graphic t test ($P = 0.05$). Softening of the lateral

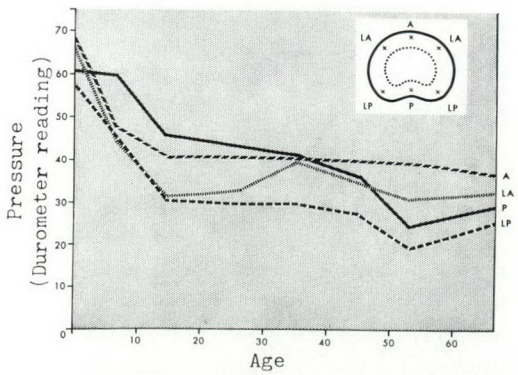

Fig. 11.—Pressure variation on the annulus.

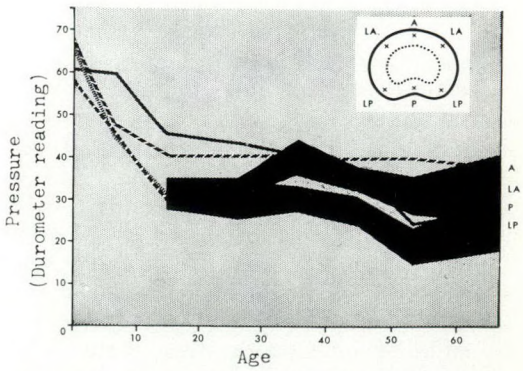

Fig. 12.—Wide bands indicate four times the standard error of mean value.

posterior fibres becomes statistically significant at 32 years of age and these continue as the weakest area of the entire annulus until about 48 years when the posterior lamellae degenerate.

Softening occurs in all regions in the sixth decade. The slight terminal increase in most regions is due to thinning and atrophy of the annulus, which permit distortion of the pressure gauge readings by the underlying bone surfaces.

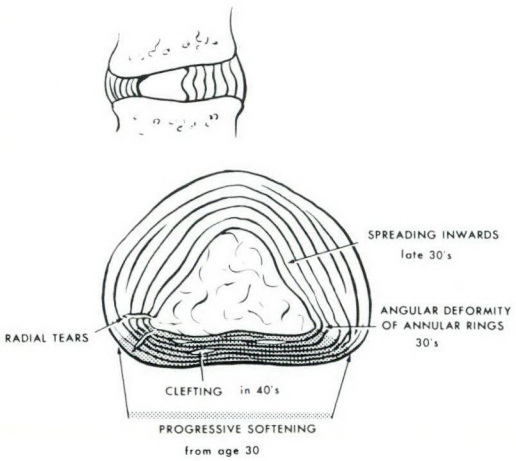

Fig. 13.—Diagrammatic chronology of the destructive process.

Discussion (Fig. 13)

While the nucleus is in a gel or semi-gel state, it must follow the mechanical laws of a contained viscous fluid: (a) because it is essentially non-compressible, any force that changes its shape will change the shape of the container (annulus or cartilage plate); (b) pressure at any point in the fluid remains equal; and (c) displacement of the fluid and container will take place first in the area of least resistance.

The intervertebral disc begins to show (degenerative?) changes as early as 11 years of age when embryologic vascular channels are reopened, disrupting the avascular gel state of the nucleus pulposus. By this time, however, the lumbar spine has for some years established its adult type of lordotic curve. The suggested sequence of events is as follows: As the lordotic curve and erect stance develop, the child, when standing, frequently arches his back to the extent that the posterior fibres of the annulus become weight-bearing components rather than the limiting membrane of a fluid pressure system. This stress increases in force, duration and frequency as the child develops. In the early stages the nucleus still exerts centrifugal pressure while weight bearing; thus the posterior fibres are compressed together and forced to bulge outwards simultaneously. As the posterior annulus gradually takes more weight, however, the nucleus is called upon to take less and the tendency to bulge centrifugally progressively diminishes. As the weight-bearing centre migrates posteriorly onto the compressed annulus, the repeated stretching of the anterior annular fibres by the diverging vertebral surfaces tends to straighten these hitherto centrifugally arched annular rings. This allows them eventually to bend inwards as the nucleus gradually loses its function of pressure transmitter.

By the time there is a significant weakening at the lateral posterior corners of the annulus, the nuclear tissue has undergone alteration from a gel to a more fixed fibrous state. The total nuclear volume decreases thereafter by dry necrotic degeneration within the fibrous nucleus.

Posterior and lateral posterior annular fibres of the disc undergo a gradual pressure atrophy,[4] with a loss of much of their volume. The particular combination of twisting and atrophy of the lateral posterior lamellae makes this area especially vulnerable to early clefting and radial tears.[5-7]

When compression forces are applied to intact intervertebral discs, a breakdown may occur in the posterior regions of the joint, producing a radial tear.[8, 9] Study of these events demonstrates the immediate pathology of disc rupture as applied to symptomatic disease. This paper describes the pathophysiological changes that render the posterior areas prone to such ruptures and shows that radial tears occur also as an asymptomatic ageing process. Thus, a radial tear must be viewed in the light of the normal variations in the disc with age. For symptoms to arise from the disc it is probable that nuclear and/or annular tissue must protrude to such a degree as to impinge upon the sensitive peripheral annular ring.

Summary and Conclusions

Degeneration of the lumbar intervertebral disc follows a definite chronological order beginning early in the second decade. The mechanism by which the lateral posterior corners of the annular lamellae become the weakest area of the entire annulus is described.

This weakening becomes statistically significant at 32 years of age in the "normal" disc. Annular clefting predisposes to complete lateral posterior radial tears, and later to large direct posterior tears.

When the nuclear and posterior annular tissue has degenerated, progressive changes towards an ultimate fibrous ankylosis of the intervertebral joint proceed.

The changes described are compatible with normal function, but should they be considered as normal ageing? Degenerative disc disease is endemic to modern society,[10] where apparently the lumbar disc, as a result of lordosis, is subjected to mechanical stresses which are greater in its posterior portion. The limitation of the pathologic changes to the area of the disc subjected to these concentrated pressures suggests that the changes are not properly characterized by the term "degeneration" but are destructive changes resulting from trauma (Fig. 13). It is suggested that the trauma concerned is that of intermittent and prolonged pressure, causing a pressure fatigue of the posterior portion of the annulus, becoming measurable in the early twenties and proceeding through the successive stages of softening, clefting and radial tears to general destruction of the posterior quadrant. In the advanced case the pressures are finally transferred forward to the remainder of the disc and complete destruction is the result.

Thanks are extended to Dr. S. M. Friedman, Professor, Department of Anatomy, University of British Columbia; Mr. C. G. Lemon, Department of Anatomy; the Department of Medical Illustration; Dr. Donald O. Anderson, Associate Professor, Department of Preventive Medicine and Mrs. Patricia Ritchie.

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Résumé

Nous avons étudié, au point de vue morphologique, le disque intervertébral. Il en ressort qu'il existe une relation de cause à effet entre l'apparition des modifications dégénératives qu'on observe et les tensions physiques dont est l'objet le patient atteint de lordose.

La dissection a permis de montrer que le processus de destruction commence vers 48 ans, mais qu'il est précédé par un ramollissement progressif au même niveau, qui débute vers l'âge de 30 ans et qui s'aggrave lentement jusqu'au moment où apparaissent les signes de destruction. Les modifications dégénératives sont alors observées dans la même région.

Le mode d'évolution de la vie du disque intervertébral permet de croire qu'il est indiqué de corriger "en profondeur" la tension physique de la lordose lombaire pour traiter la dégénérescence discale.

STUDY OF GASTRONE-LIKE ACTIVITY OF INTESTINAL SECRETION IN THE ISOLATED PERFUSED CANINE STOMACH*

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GASTRIC - secretion - inhibitory activity of gastric juice, attributed to the agent "gastrone", was first reported by Brunschwig *et al.*,[1-5] who found that the intravenous injection of preparations of gastric juice inhibited gastric secretion in pouch dogs. Normal and achlorhydric human gastric juices were tested and techniques for assay were suggested. More precise quantitative methods for assaying gastrone were developed by the researchers of the Mayo Foundation, Rochester,[6-12] who confirmed the work of Brunschwig's group.

An interesting aspect of the work on gastrone was the production of achlorhydria and gastric mucosal atrophy in dogs subjected to repeated injections of dialyzed and lyophilized human gastric juice.[13-16] However, when Sircus *et al.*[17] tried to reproduce the experiments of Smith *et al.*,[15] they were unable to induce gastric atrophy in dogs using extracts of human gastric juice. More recently rats have been used to assay the gastrone-like activity of human and canine gastric juice.[18-22]

The source of gastrone has not been clearly determined. The most acceptable suggestion is that the inhibitory substance present in gastric juice is secreted primarily by the pre-pyloric glands.[11-23] The richest known source of gastrone is achlorhydric human gastric juice.[12] The mechanism of gastrone activity remains uncertain. It does not seem to interfere with parietal cell function nor does it alter the luminal-mucosal barrier to insorption.[24]

Gastrone-like activity has also been found in saliva,[25] thoracic duct lymph[26] and intestinal secretions.[27] Rudick *et al.*[27] studied extracts of dialyzed and lyophilized secretions from canine intestinal pouches. When these were injected into pouch dogs and pylorus-ligated rats, significant inhibition of gastric secretion was observed. These investigators have postulated that a gastrone-like inhibitor is present in both jejunal and ileal secretions. Some components of intestinal secretions have shown marked biochemical and pharmacological similarity to the gastrone derived from antral secretions.[27]

A review of the literature suggests that a cause for disagreement between various investigators who have studied gastrone-like activity relates to the differences in both preparation of extracts and in methods of assaying them.[12] The pylorus-ligated stomach, with surgically induced hyperacidity, is not an ideal preparation for the assay of a secretory inhibitor.[28] The use of a Heidenhain pouch, which is really not a totally denervated preparation, also presents some disadvantages when the stimulatory or inhibitory characteristic of a "humoral factor" is assayed.[29]

In our experience some of the difficulties that have been encountered in studies carried out using *in vivo* gastric pouches in dogs can be avoided by using a homologous *ex vivo* perfused canine stomach.[29-32] The purpose of the present investigation was to study the possible gastrone-like activity of dialyzed, lyophilized extracts from canine jejunal and ileal secretions. These extracts were assayed using histamine-stimulated isolated canine stomachs.

METHODS

Perfusion System

The methods and an apparatus (Fig. 1) designed to study the isolated perfused stomach were developed in our laboratory. A detailed description of our perfusion system has recently been published.[33] In the process of developing our stomach perfusion technique the importance of the following conditions became apparent: (a) rigid maintenance of hemodynamic and physical parameters, (b) preservation of normal venous pressure dynamics, and (c)

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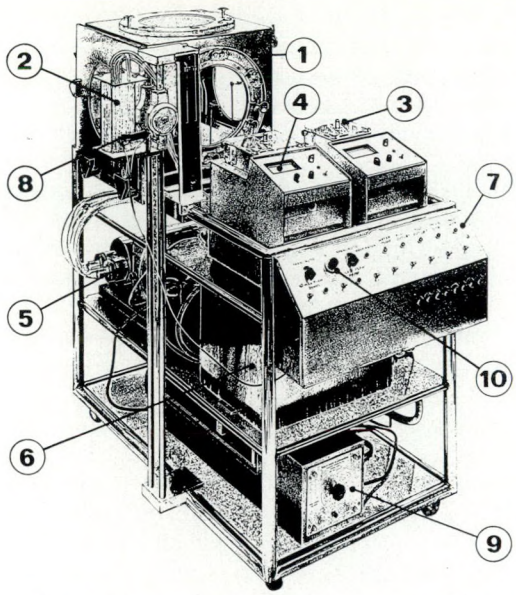

Fig. 1.—Apparatus designed for the study of the isolated perfused canine stomach: 1—perfusion chamber, 2—venous reservoir, 3—arterial pump, 4—venous pump, 5—respirator pump, 6—water bath, 7—electronic control panel, 8—photocell for venous pump control, 9—infusion pump, 10—arterial pressure selector for arterial pump control.

the use of a supporting animal to provide the necessary perfusate and to serve also as a physiological dialyzer and oxygenator. The first two conditions are automatically provided for by our perfusion apparatus (Fig. 1).

The principal component of our apparatus is an organ perfusion chamber which assures a precise control of temperature, humidity and pressure surrounding the isolated stomach. Specially designed cannulas permit rapid connection of the organ to the perfusion circuit and allow for the measurement of pressures within the main vessels of the organ.[33] The blood-flow circuitry is shown schematically in Fig. 2. Blood is pumped to the organ by an occlusive roller pump (Sarns low-flow perfusion pump No. 5M 6050, Travenol Laboratories Ltd., Alliston, Ont.). Arterial blood-flow rate can be arbitrarily varied by adjusting the pump speed selector or it can be controlled automatically to deliver a constant selected arterial perfusion pressure. The latter facility utilizes a feedback circuit which responds to the galvanometer voltage from an arterial pressure-channel on a biomedical recorder (Offner Type R biomedical recorder, Beckman Instruments Inc., Palo Alto, Calif.). Venous outflow from the organ is conducted to a height-adjustable reservoir located near the chamber door. A second roller pump (Sarns modular pump No. 5M 6002) returns blood to the supporting animal at a rate that is automatically controlled by photoelectric sensing of the blood level in a sealed venous reservoir. Because perfect hemostasis is seldom achieved in the stomach preparation, a

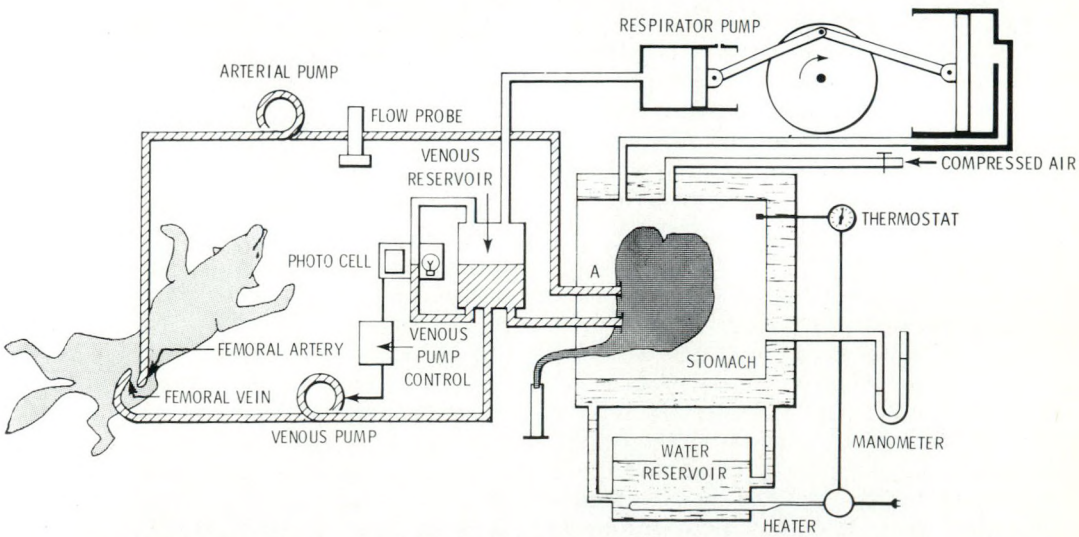

Fig. 2.—Circuitry for *ex vivo* homologous perfusion of the canine stomach.

small amount of blood escapes from the flow circuit within the chamber. A third pump delivers this blood by the main venous return line to the supporting animal. Three-sixteenth-inch tubing is used to conduct extracorporeal blood flow. The entire flow circuit has a priming volume of 200 ml.

Supporting Animal

For prolonged perfusions a method of controlling the normal metabolic properties of the blood is desirable. At present the most suitable way of providing for this is to use a whole animal to support the isolated organ.[28-33] The supporting animals used in this series were prepared by total gastrectomy and end-to-end esophagoduodenostomy which was carried out 7 to 21 days before stomach perfusion.

In preparation for stomach perfusion the supporting animals were anesthetized with 15 mg./kg. body weight of sodium pentobarbital (Nembutal). They also received an antihistamine, promethazine hydrochloride (Phenergan, Poulenc Ltd., Montreal, Que.) in a dose of 50 mg. at the beginning of anesthesia.

During perfusions the supporting animals breathed spontaneously, but intermittent positive-pressure hyperinflation was carried out and endotracheal oxygen insufflation was provided if arterial Po_2 was less than 60 mm. Hg. Physiological saline was administered to the supporting animal at the rate necessary to maintain stable half-hourly hematocrit determinations. Systemic arterial pressure was continuously monitored and rectal temperature was measured. Normothermia in the supporting animal was maintained using an adjustable heating blanket.

Stomach for Perfusion

Mongrel dogs, weighing 15 to 25 kg., provided the stomachs which were studied. Following a 24-hour fast, the stomach donor was anesthetized, using 60 mg./kg. Nembutal. Following endotracheal intubation the donor was placed in the supine position and peripheral cut-downs were performed for the monitoring of systemic arterial pressure and the administration of intravenous fluids.

Through a long midline laparotomy the stomach was prepared, carrying out the following sequence of procedures: (1) splenectomy, (2) pancreatectomy, (3) enterocolectomy (proximal duodenum to sigmoid), (4) left nephrectomy, (5) division of the common bile duct, (6) mobilization of the hepatic artery and portal vein, (7) division of the lesser omentum, (8) median sternotomy, (9) division of the diaphragm in the midline, (10) division of the lower thoracic esophagus, and (11) mobilization of the lower thoracic aorta.

A transpyloric secretion-drainage cannula was introduced and advanced to the level of the distal stomach-body. The animal was then given 3 mg./kg. of heparin as an anticoagulant and the following procedures were rapidly carried out: (1) ligation and division of the infraceliac aorta, (2) division of the thoracic aorta, (3) division of the retroperitoneal attachments of the paraceliac aorta, and (4) division of the distal portal vein.

Control of Perfusion

Isolated stomachs with their entire vasculature preserved were placed on a tray in the perfusion chamber so as to rest on their ventral wall. A moderate inclination in the gastric body directed secretions to the drainage cannula. The stomach vasculature was connected to the extracorporeal circuit by the arterial cannula in the pre-celiac segment of the aorta and the venous cannula in the distal portal vein. The chamber was then sealed and intermittent, respiratory-rate pressures of +8/+3 mm. Hg were produced in the chamber by one of two reciprocally driven respiratory pumps. The second pump synchronously produced intermittent negative pressures in the sealed venous reservoir. The reservoir height and the pump volumes were adjusted to produce pressures of +6/+9 mm. Hg in the portal vein. These cyclic variations in the chamber and portal venous pressures represent an attempt to simulate the effects of normal respiration on intra-abdominal pressure and gastric venous-pressure dynamics. The arterial perfusion pressure was controlled at 110 mm. Hg.

When the aforementioned parameters

had become stabilized, intra-arterial infusion of histamine was begun.

Measurements

In the present study the arterial and portal venous pressures and the arterial flow rate (Zepeda EPD-2RD flowmeter, Zepeda Instruments, Seattle, Wash.) were continuously monitored. Arterial and venous blood gases and oxygen contents were measured hourly. Stomach oxygen consumption was calculated, using the arterial flow rate and the arteriovenous oxygen content differences which were measured by the Van Slyke method. The volume, pH, total HCl (titrable to pH 7.0), sodium, chloride and pepsin of half-hourly collections of secretion were measured.[32]

Extracts of Intestinal Juice

Secreting jejunal or ileal pouches were produced in mongrel dogs weighing 15 to 25 kg. In three dogs, a pouch of the second 15 cm. of jejunum was constructed, and in three, the second last 15 cm. of ileum was used for the secreting pouch.

In all dogs the segment to be used for the pouch was divided with its vasculature intact, and the proximal and distal ends of the segments were closed by inverting sutures. Intestinal continuity was restored by end-to-end anastomosis of the divided bowel. A stainless steel secretion drainage cannula was introduced at the antimesenteric border of the isolated loop and the cannula with attached pouch was secured to the peritoneum at the site of a stab wound through which the drainage cannula was passed.

Collection of the pouch secretions began seven days after operation. Twenty-four-hour collections were made from each animal while the dogs received regular daily meals. The 24-hour collections from the three pouch dogs in each group were pooled, diluted in distilled water (1:2) and refrigerated until a sufficient amount was available for dialysis.

The pooled diluted secretions were then filtered through glass wool and dialyzed with a cellophane membrane dialyzer at 5° C. against distilled water. The rate of dialysis was 8 ml. of diluted secretion per minute. The products of dialysis were refiltered and separate lyophilization of the supernatant and precipitate was carried out. The final products in the form of dry powder were weighed, bottled under vacuum and stored in a freezer until use. One litre of pooled jejunal juice produced 7.9 g. of extract and 1 l. of ileal juice provided 7.3 g. of extract.

Assay of Intestinal Extracts

Immediately before infusion the extracts were dissolved in normal saline at pH 7.0. When a relatively steady rate of acid secretion by the *ex vivo* stomach had been achieved, intra-arterial infusion of the extract was begun. In this way the extract was delivered directly to the isolated stomach. The hourly rate of extract infusion was calculated on the basis of the body weight of the supporting dog. In all experiments the extract infusion rate during the first hour was 2 mg./kg. body weight. In the next hour the rate was increased to 4 mg./kg. The infusion of extract was then interrupted for a period of one to two hours. It was then resumed for two hours at increased rates of 6 mg./kg./hr. and 12 mg./kg./hr. for the third and fourth hour of extract infusion, respectively. Every experiment lasted a minimum of nine hours and during this period the extract was infused for two two-hour periods. Relatively large doses of extract were used in these experiments in an attempt to induce a reproducible alteration in the secretory activity of the histamine-stimulated isolated stomachs.

Eight experiments were carried out to assay the effects on gastric secretions. In four experiments the jejunal extracts were studied and in four the ileal extracts were infused. Four days before stomach perfusion two supporting dogs for the jejunal extract series and two for the ileal extract series were prepared by total jejunectomy or total ileectomy, respectively.

Results

Histamine Stimulation

Figs. 3 and 4 summarize the reactions of two *ex vivo* canine stomachs which were stimulated by sustained intra-arterial

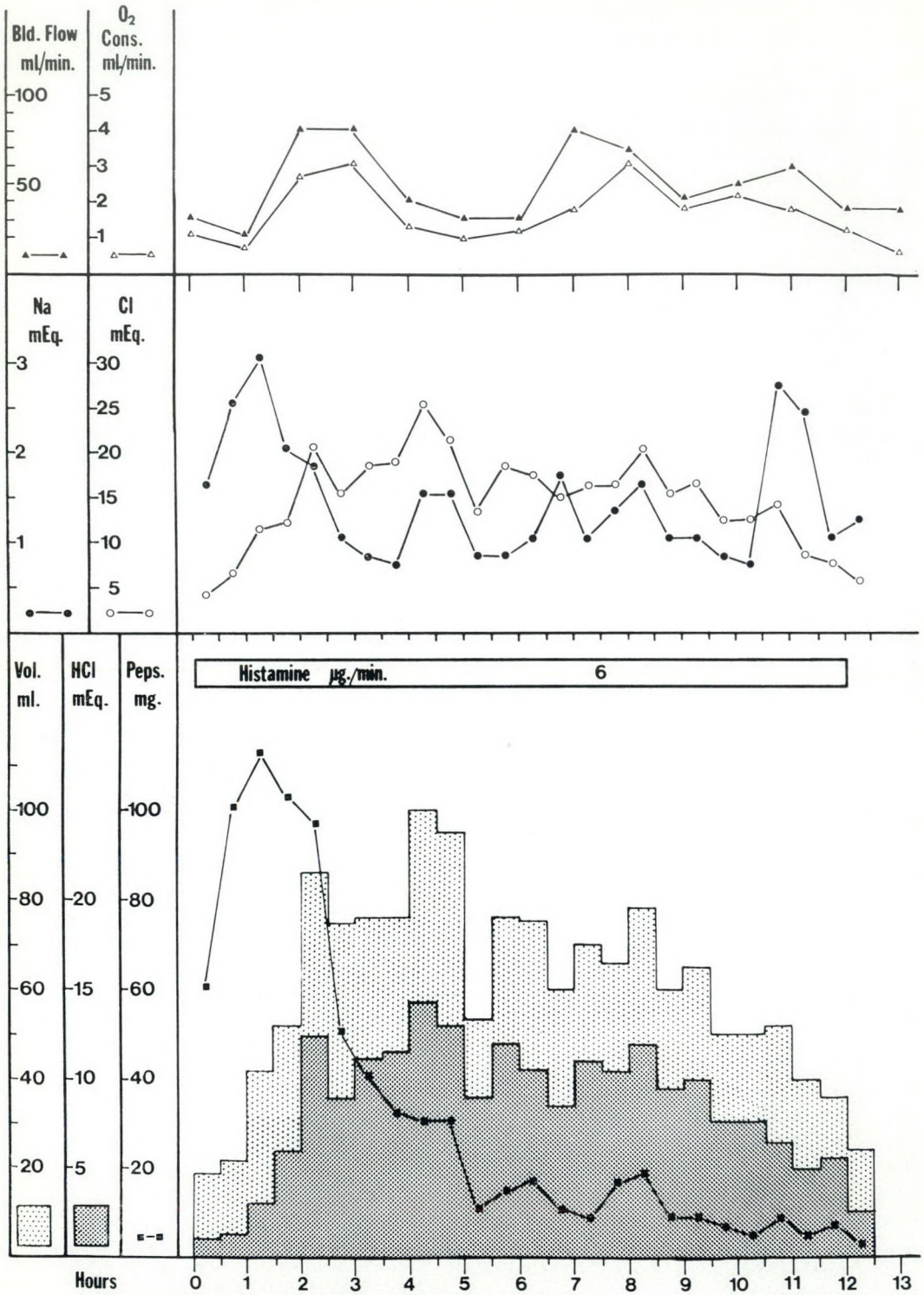

Fig. 3.—Control histamine-stimulated stomach. Donor dog, 12 kg.; supporting dog, 20 kg. 30-minute output of gastric juice; volume, total HCl, pepsin and electrolytes. Hourly measurements of blood flow and oxygen consumption (see also Figs. 4 to 8).

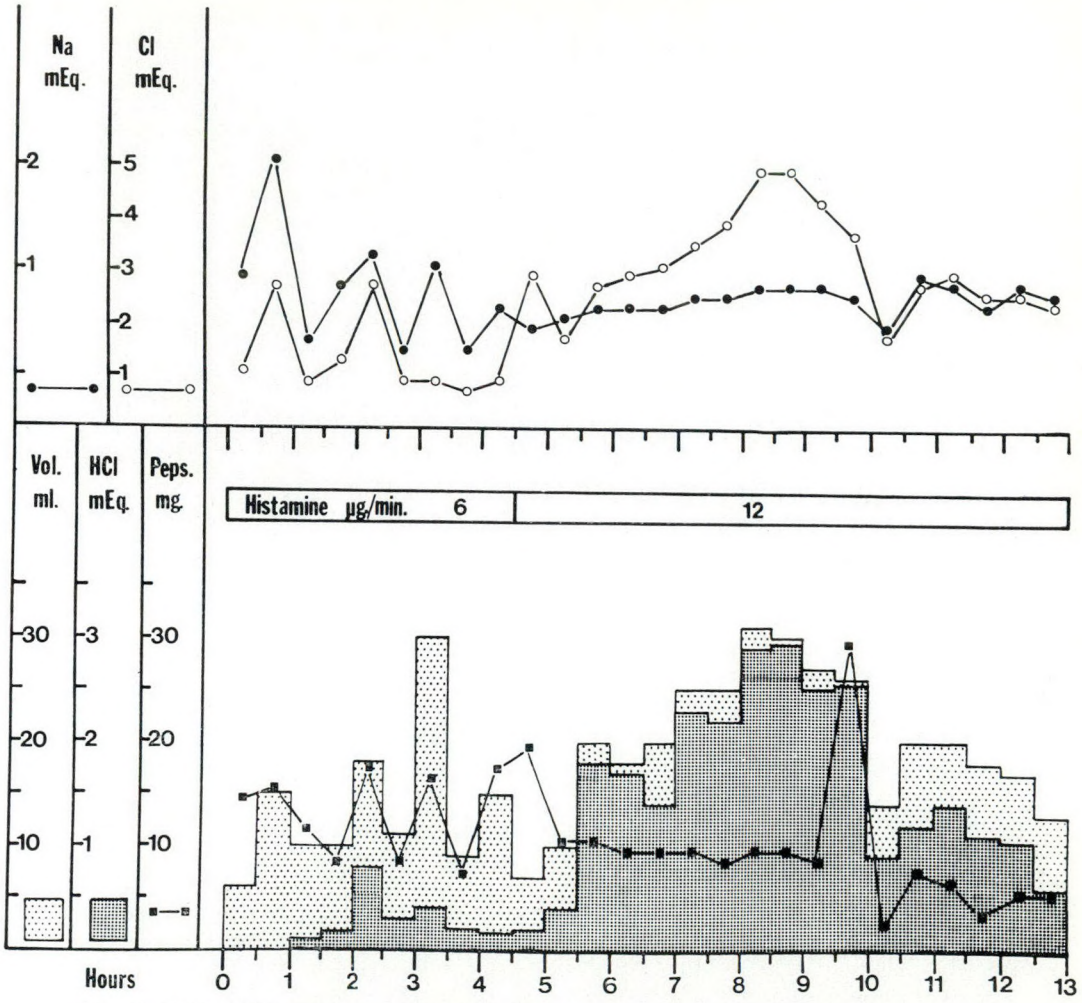

Fig. 4.—Control histamine-stimulated stomach. Donor dog, 15 kg.; supporting dog, 13 kg.

infusion of histamine. These two experiments were representative examples from a series of seven control stomach perfusions.

The secretory response of these stomachs compares well with our previous reports.[29-32] The stomach whose results are shown in Fig. 3 produced a large quantity of acidic juice and the rate of secretion was relatively constant for more than 10 hours. An identical dose of histamine in another stomach (Fig. 4) produced a secretory response which was somewhat delayed and less pronounced. When the dose of stimulant was doubled, secretion improved but the response was not prolonged.

The gastric secretory response to histamine varies considerably from preparation to preparation and a perfectly reproducible pattern of response cannot be achieved. This difficulty must be appreciated when attempts are made to assay factors that are thought to alter gastric secretory functions.

Extracts of Jejunal and Ileal Juices

None of the eight extract-infusion experiments demonstrated an "acceptable" inhibition of gastric secretion. Acceptable inhibition was considered to be a decrease in secretion that could be related to the period when extract was infused and inhibition of a magnitude comparable to that reported by Rudick *et al.*[27] who observed a 90% reduction in secretion follow-

TABLE I.—Effect of Infusion of Extracts from Intestinal Juice on Volume and Total HCl Output of *Ex Vivo* Canine Stomachs. Results Expressed in Percentage of Changes from "Basic Values" (Volume and HCl Output Obtained During 30 Minutes Immediately Before the Beginning of Infusion). Eight Stomachs Perfused.

Averages (Av.), Standard Deviation (SD) and Range, with Smallest (S) and Largest (L) Values

Continuous histamine stimulation

30-minute periods		*Extract**				*No extract*		*Extract**				*No extract*	
		2		*4*				*6*		*12*			
†													
Volume	Av.	+17	+27	+35	+37	+40	+37	+53	+69	+61	+54	+42	+36
	±SD	18	25	42	51	55	69	69	79	75	90	73	72
	S	0	0	−13	−11	−20	−20	−19	−20	−24	−28	−28	−28
	L	+46	+60	+97	+114	+114	+200	+200	+220	+250	+119	+190	+190
HCl output	Av.	+30	+62	+74	+59	+39	+56	+93	+117	+104	+89	+83	+54
	±SD	26	46	72	73	61	84	105	131	125	122	143	111
	S	0	+10	−8	−10	−14	−10	−22	−22	−24	−25	−23	−10
	L	+80	+140	+153	+190	+135	+240	+290	+350	+290	+290	+390	+260

*Dosage of extract (2, 4, 6 and 12) in mg./kg. body weight of supporting dog per hour.

†Interrupted line means that not all 30-minute periods, preceding or following extract infusion, are recorded. The excluded "periods" were not relevant for evaluation of effect of extracts.

ing jejunal extract and a 75% reduction following ileal extract during histamine stimulation. The response of eight stomachs to the infusion of extracts of intestinal juice is summarized in Table I. Changes occurring in gastric secretion (volume and output of total HCl) during and after perfusion of extract are expressed in percentage difference from "basic values". Volume and HCl output obtained during the 30-minute period immediately preceding the beginning of extract infusion are considered "basic values". There is no evidence from this table that the extracts used merit the name of "inhibitors". Figs. 5 and 6 show the results in two stomachs that were subjected to the action of jejunal extracts and Figs. 7 and 8 the effect on two stomachs into which ileal extracts were infused. Certainly none of these experiments demonstrated acceptable inhibition of acid secretion for, indeed, the secretory responses compare very closely to those of the two control experiments (Figs. 3 and 4).

A careful study of the blood-flow rate and oxygen consumption as well as of the sodium and chloride measured in the secretions also failed to demonstrate any relationship between changes in these measurements and the infusion of the extracts. As expected, however, variations in these values are related to the secretory response to histamine.

Discussion

The infusion of exogenous histamine is necessary to maintain basal secretion in our *ex vivo* isolated stomach preparation. No direct relationship has been found between the dose of histamine and the secretory response of the stomach, nor can the magnitude of the secretory response be related to the weight of the donor dog. It is known that the concentration of histamine in the blood is not related directly to the secretory response of parietal cells.[34] The response to histamine depends upon the reactivity of parietal cells at a given moment and upon the number of parietal cells that are stimulated. Sustained histamine stimulation does not produce a prolonged secretory response and an ideal plateau rate of secretion does not last more than a few hours. Parietal cells likely become "accustomed" to the stimulant and in addition, under conditions of prolonged stimulation, "exhaustion" of these cells undoubtedly occurs. The mucosa of the isolated stomach is vulnerable and changes in conditions of the perfusion system are also likely to affect the secretory activity of the parietal cells.

Notwithstanding these considerations, the isolated stomach preparation appears reasonably suitable for the assay of humoral inhibitory factors which are thought to have an immediate effect on gastric secretion. The preparation is totally

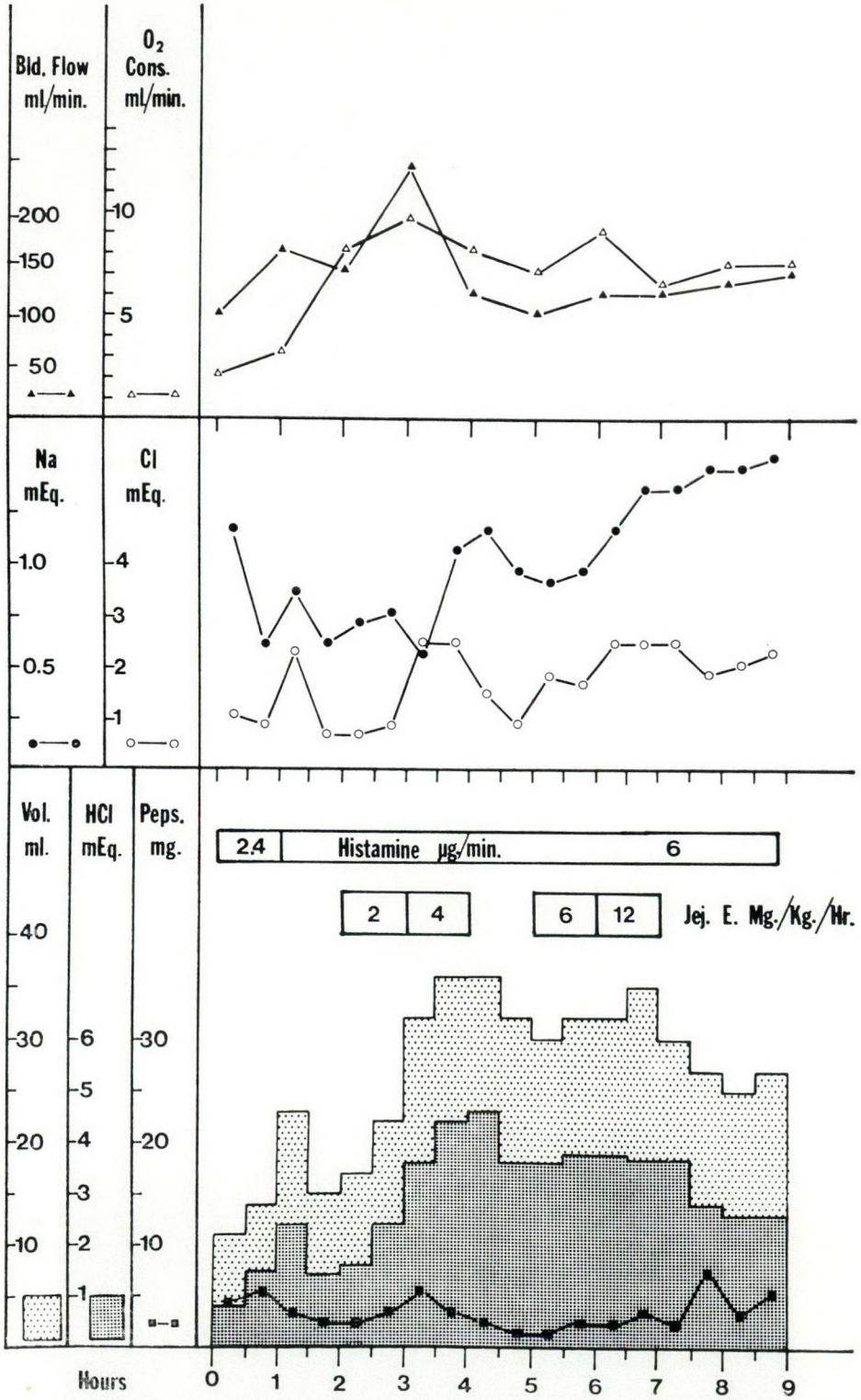

Fig. 5.—Assay for jejunal extract (Jej. E.). Donor dog, 17 kg.; supporting jejunectomized dog, 28 kg.

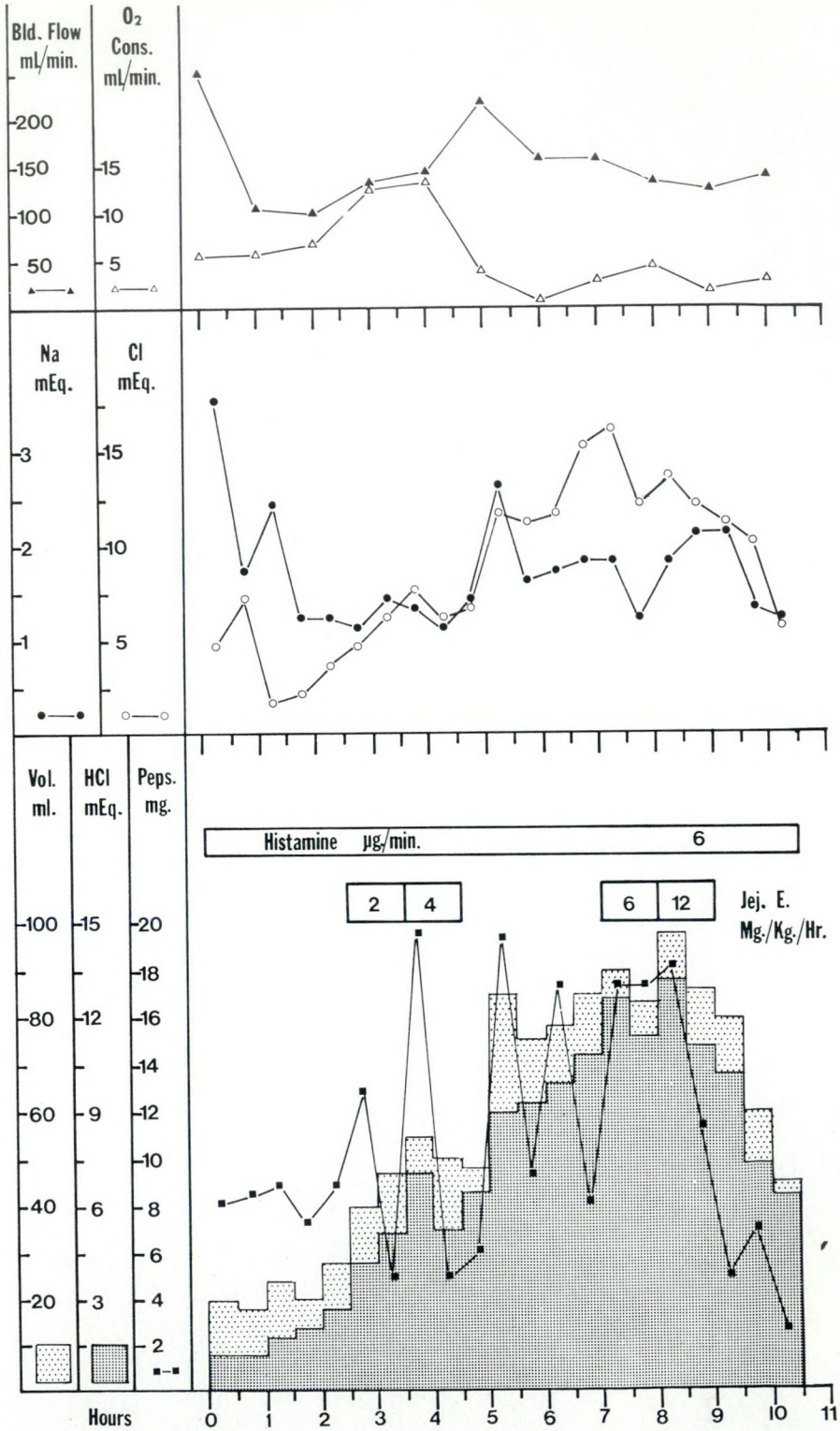

Fig. 6.—Assay for jejunal extract. Donor dog, 15 kg.; supporting dog, 20 kg.

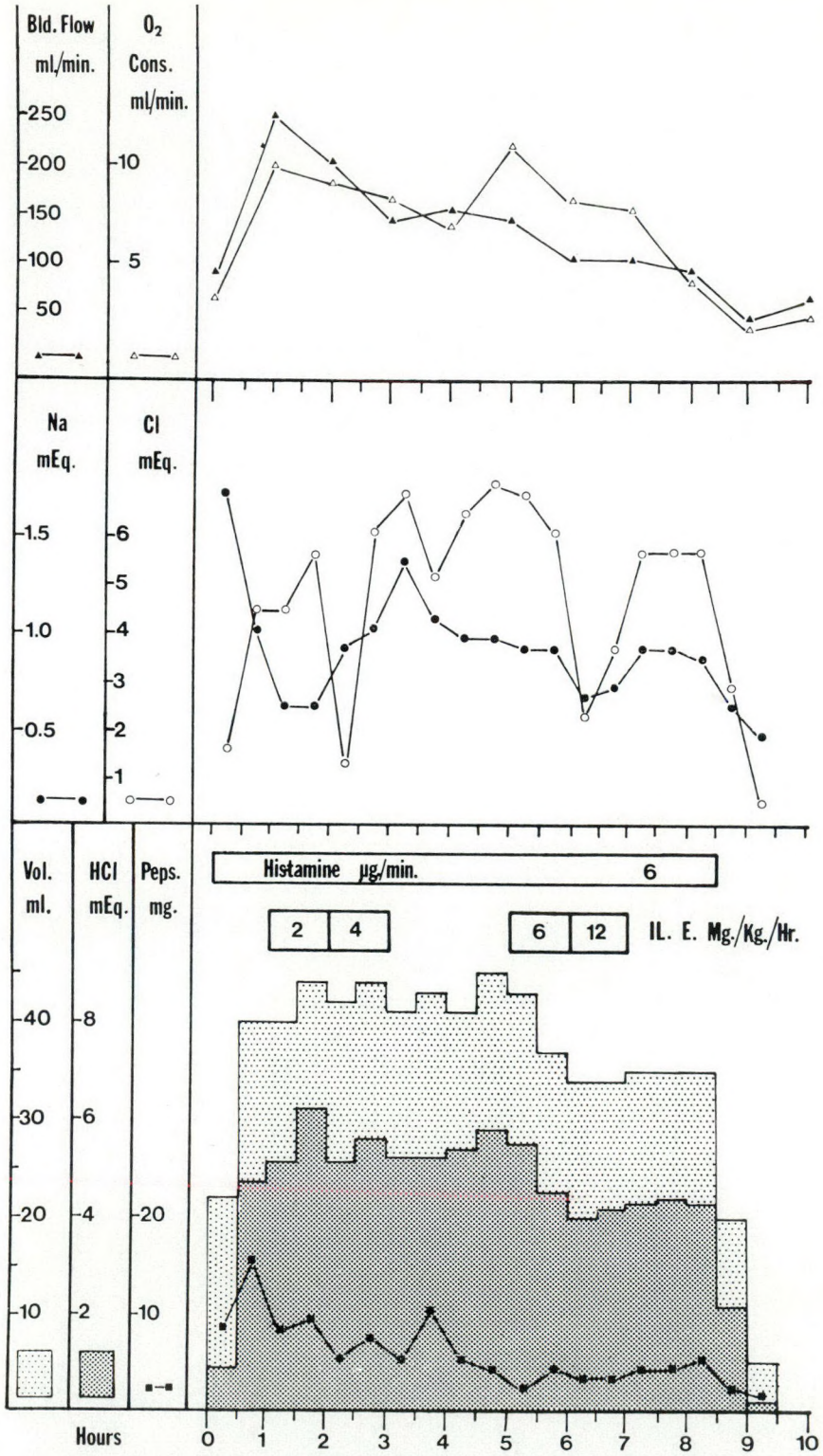

Fig. 7.—Assay for ileal extract (Il. E.). Donor dog, 9 kg.; supporting dog, 17 kg.

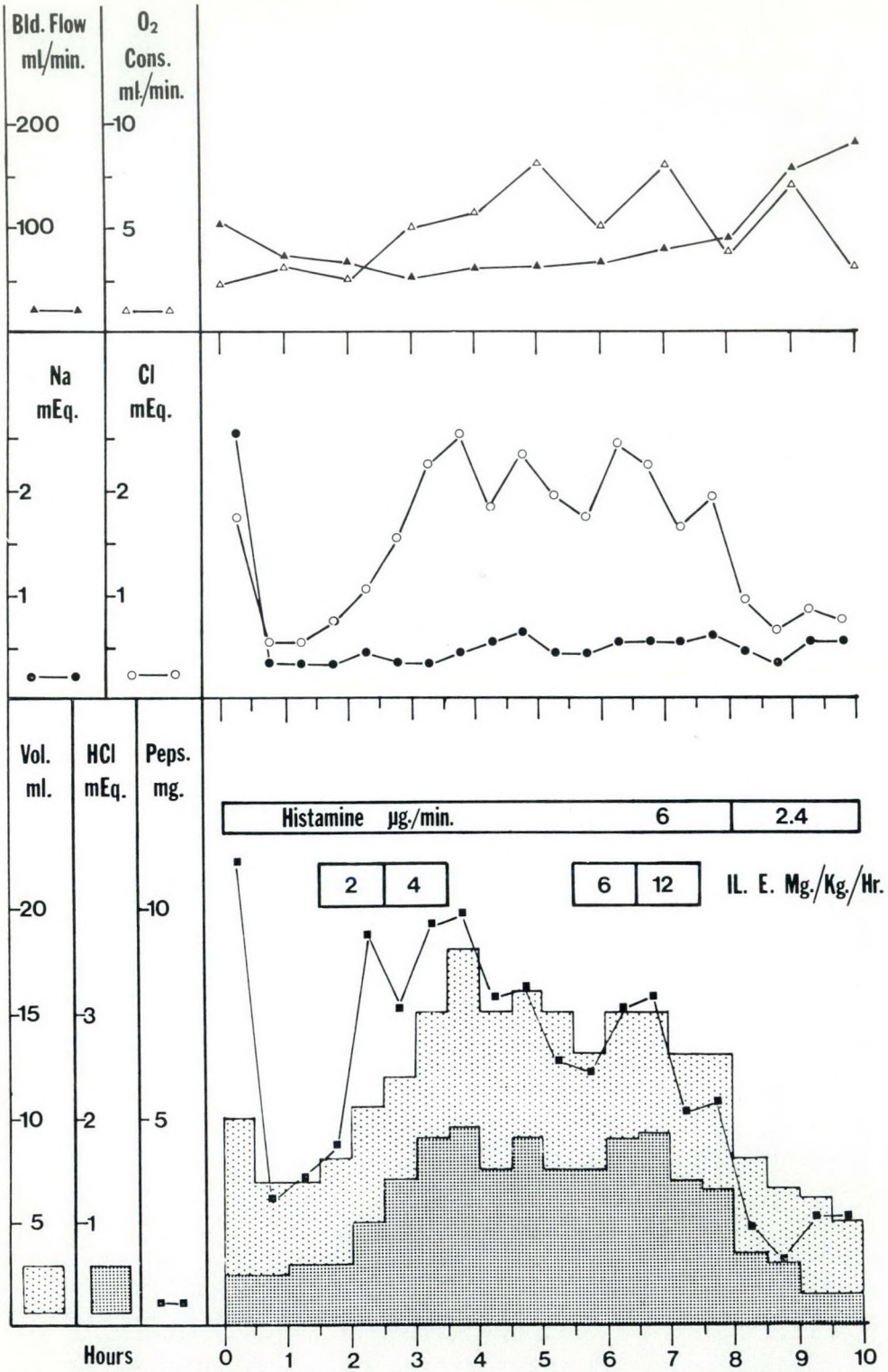

Fig. 8.—Assay for ileal extract. Donor dog, 18 kg.; supporting ileectomized dog, 19 kg.

denervated, its circulation can be well controlled and the secretory function can be precisely evaluated. A stimulant or inhibitor can be delivered directly to the gastric mucosa by the arterial system and, under these conditions, a true inhibitor should produce a detectable alteration in secretory activity of the isolated organ.

What features characterize a true gastric secretory inhibitor? The literature has not provided a definition of the requirements in this regard. Certain reports dealing with inhibitors, however, specify that acceptable inhibitors have a relatively rapid and reproducible effect on gastric-juice production when they are administered during a "plateau period" of gastric secretion. For example, the output of HCl from Heidenhain pouches in dogs was inhibited by as much as 50% to 75% within 20 to 45 minutes of intravenous administration of secretin,[35] hog gastrin[36] or cholecystokinin.[37, 38]

The effective doses of inhibitory substances have differed in various studies. Smith *et al.*,[15] in their original study of the extracts from human gastric juice, which were assayed on Heidenhain-pouch dogs, found that a maximal inhibition was produced by 1.0 mg. of extract per kg. body weight. Sircus *et al.*,[17] using Smith's methods, did not observe inhibitory action by human gastric juice extract when doses of up to 2 mg. of extract per kg. body weight were used. Extracts of gastric secretions from dogs when assayed in rats[24] were found to be effective in a dose of 5 mg./kg. Rats tolerated doses 10 times larger than the "effective dose".[24] Jejunal extracts from intestinal pouch secretions of dogs were found to be effective inhibitors in pylorus-ligated rats in a dose of 10.8 mg./kg. body weight.[27] The dose necessary for gastric inhibition in rats is apparently much higher than it is in dogs.

Marked inhibition of gastric secretion by intestinal extract has been reported by Rudick *et al.*[27] In their studies, 1 mg./kg. of jejunal or ileal extract was injected intravenously over a period of 30 minutes into the animal from which the extract had been derived. Acid output by histamine-stimulated Heidenhain pouches was reduced by 90% and 75% respectively. The report of these results, however, did not mention the time of appearance nor the duration of the apparent inhibitory response. Additional information provided by Rudick,[39] reveals that 60% of the inhibition in these studies occurred in the first hour following injection of extract but maximal inhibition did not take place until the second hour.

Our experiments cannot be directly compared with those of Rudick.[39] We have used pooled intestinal juice. The functioning isolated stomachs were obtained from animals that did not contribute to the collection of intestinal secretions. Furthermore, we injected the extract directly into the arterial system of the stomach and therefore the extracts reached the organ in high concentration. Our hourly dose was much higher than those used by Rudick *et al.*[27] and the duration of extract infusion was longer. Under the conditions of our study we were not able to induce secretory inhibition in the *ex vivo* stomachs, nor were we able to observe any changes in the stomach blood flow, oxygen consumption or electrolyte composition of the gastric juice.

The problem of "gastrone-like substances" has not been satisfactorily solved. Sircus *et al.*[17] were unable to reproduce the classical experiments of Smith *et al.*[15] with respect to the inhibitory action of human gastric juice on the secretions of Heidenhain-pouch dogs. Rudick recently stated[39] that he occasionally observed pyrogenic reactions in the dog in which he was assaying intestinal extracts.[27] A pyrogenic reaction may be a complicating factor in the evaluation of gastrone-like activity of relatively crude intestinal extracts.[39] Sircus *et al.*[17] discussed the possibility that some "positive reactions" to "gastrone-like extracts" may be due to antigen-antibody responses which induce a secondary alteration in the activity of the parietal cells. In our experiments no pyrogenic reaction to the extract was observed in the supporting dogs. Before subjecting our isolated stomach preparation to infusions of extract, we carried out a series of four pilot experiments wherein the extract was infused intravenously in

large doses into intact dogs, and the cardiorespiratory status of the recipient animal was studied. No disturbance in blood pressure, heart rate or body temperature was provoked in these dogs. There has been no evidence that the intestinal extracts contained a sufficient amount of antigen[17] to inhibit the parietal cells in the isolated canine gastric mucosa. Our failure to confirm the "gastrone-like" activity of intestinal extract under the described conditions of the experiments, should stimulate further studies which will utilize more purified extracts.

Summary and Conclusions

Lyophilized extracts of canine jejunal and ileal secretions were assayed for possible gastric inhibitory action on the *ex vivo* isolated canine stomach. Other authors have found that similar extracts significantly reduce acid production in histamine-stimulated canine Heidenhain pouches. In contrast to previous reports, we have found that intestinal extracts, when infused intra-arterially, do not inhibit gastric secretion in the isolated stomach. An *ex vivo* canine stomach preparation is described in detail and the advantages of such a preparation for the study of gastric physiology are discussed.

The technical assistance of Mr. J. McCallum, Mr. R. Orchard, Mrs. M. McCubbin and Mrs. D. Emerson is gratefully acknowledged.

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Résumé

L'objet de cet article était d'étudier l'activité gastronoïde possible d'extraits de sécrétions intestinales du chien (jéjunum et iléum). Ces extraits ont été essayés sur des estomacs de chien stimulés par l'histamine.

Durant la période de perfusion, d'une durée de 9 à 10 heures, divers paramètres ont été étudiés continuellement: les pressions artérielle et veineuse et la vitesse de la circulation artérielle; les gaz sanguins artériel et veineux toutes les heures; le calcul de la consommation d'oxygène de l'estomac a été basé sur la vitesse de circulation artérielle et sur les différences de teneur en oxygène artérioveineuse, qui ont été calculées; le volume, le pH, le HCl total (titrable jusqu'au pH 7.0), le sodium, le chlorure et la pepsine des spécimens de sécrétion recueillis chaque demi-heure ont été mesurés.

Les extraits lyophilisés de sécrétions d'intestin (jéjunum et iléum) du chien ont été essayés, en vue de déterminer leur éventuelle action inhibitrice sur l'estomac isolé *ex vivo*. D'autres auteurs avaient trouvé que des extraits similaires diminuent la sécrétion acide sur des petits estomacs de Heidenhain stimulés par l'histamine. Pour notre part, nous avons constaté au contraire que les extraits intestinaux, infusés par voie intra-artérielle, n'inhibent pas la sécrétion gastrique sur l'estomac isolé. Nous décrivons en détail une préparation d'estomac *ex vivo* et soulignons les avantages que présente cette préparation pour l'étude de la physiologie gastrique.

GASTRIC SECRETAGOGUES FOLLOWING PORTACAVAL SHUNTING

It has been well established that gastric hypersecretion follows portacaval shunting in dogs, and several theories have been developed to account for this phenomenon. The leading one holds that a gastric secretagogue, normally inactivated by passage through the liver, is allowed full effect and action systemically after bypassing the liver parenchyma.

To investigate this process further, Heidenhain-pouch dogs were subjected to portacaval anastomosis by the end-to-side technique. In these dogs peripheral blood ammonia, amino acid, and histamine levels were measured as well as acid output from the gastric pouch for each of five hours after a standard meal. This feeding procedure was carried out before and after the portacaval shunt. In each instance, there was a prompt and sustained increase in gastric acid secretion. Blood ammonia levels also rose. The changes in blood amino acid and histamine levels were particularly well correlated with pouch output.

The results of this experiment suggest to the investigators that ammonia may be the specific gastric secretagogue responsible for the increase in gastric acid secretion following portacaval anastomosis.—Griffen, W. O., Slesh, M. Z. and Mooney, C. S.: Gastric secretagogues following portacaval shunting, *Surgery*, **66:** 111, 1969.

GLYCEROL AS PRIMING SOLUTION FOR EXTRACORPOREAL CIRCULATION*

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TRAUMATIC destruction of blood components, particularly red blood cells, and liberation of hemoglobin may contribute seriously to patient morbidity during extracorporeal circulation procedures of moderate or long duration. It is the purpose of this paper to report an experimental study designed to evaluate the effects of glycerol in limiting this destruction.

MATERIAL AND METHODS

Sixteen dogs varying in age, sex and weight were used. All surgical procedures were done under sodium pentobarbital anesthesia (30 mg./kg. body weight).

A vertical incision was made in the right groin. The right common femoral artery was dissected for 3 to 4 cm. below the inguinal ligament and was encircled with umbilical tape.

A right thoracotomy was then performed through the fifth intercostal space. The right lung was withdrawn posteriorly and the pericardium was opened longitudinally, well anterior to the right phrenic nerve. Umbilical tapes were used to encircle both venae cavae and purse-string sutures were placed around the right atrial appendage and wall of the right atrium. After heparinization (3 mg./kg. body weight) an arterial catheter was inserted into the right femoral artery and secured. The catheter was then attached to the arterial line of the heart-lung machine. Two vena caval catheters were inserted through the purse-string sutures in the right atrium and threaded into the inferior and superior venae cavae. These catheters were attached to the venous return line of the heart-lung machine and, when the tapes around the venae cavae were tightened, total cardiopulmonary bypass was accomplished.

A Travenol bubble oxygenator, with Mayon tubing, was used. The arterial line (¼ inch internal diameter) and venous line (⅜ inch internal diameter) were each exactly 80 inches long. A Brown heat exchanger in the outflow tract of the arterial line maintained the blood at 38° C. Pump output was maintained at 1400 ml./min. and the oxygen flow was 4 l./min. in each experimental procedure.

Two groups of experimental animals were used. Group I comprised four dogs who were subjected to total cardiopulmonary bypass for a period of three hours; the heart-lung machine was primed with Ringer's lactate solution, 20 ml./kg. body weight. Group II comprised 12 dogs also subjected to three hours of cardiopulmonary bypass, but for these animals the heart-lung machine was primed with Ringer's lactate solution containing 3% glycerol, 20 ml./kg. body weight.

At 30-minute intervals, aliquots of blood were removed from the arterial side of the oxygenator for hematocrit and plasma hemoglobin determinations. In addition, varying amounts of the priming solution were added during the course of perfusion to keep the pump output constant at 1400 ml./min.

In two dogs from Group II serial hemoglobin, hematocrit, electrolytes and blood urea nitrogen determinations were done over a period of three days.

RESULTS

As shown in Fig. 1 the average plasma hemoglobin level in Group I (Ringer's lactate prime) is higher than that in Group II (prime of 3% glycerol in Ringer's lactate). The average plasma hemoglobin level in Group I was 112 mg./100 ml. at the end of three hours, compared to 75 mg./100 ml. in Group II.

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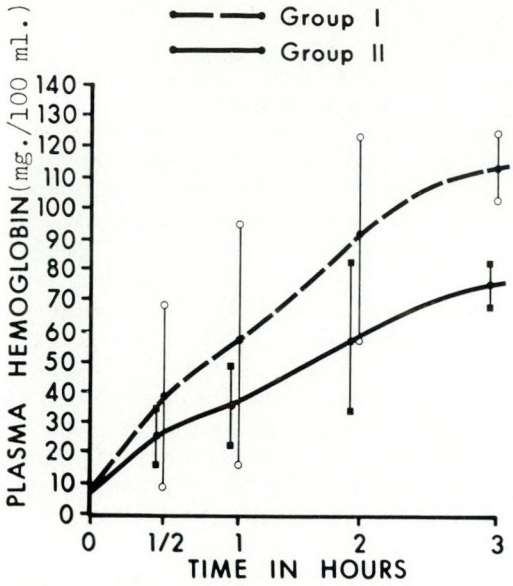

Fig. 1.—Plasma hemoglobin changes during three hours of extracorporeal bypass in Groups I and II.

Fig. 2 represents the serial hematocrits done during a three-hour period. During the first hour, very little change is noted in Group I, but a fall is observed in the dogs exposed to the glycerol priming solution. A tendency to level off is apparent after this, but in the dogs in Group II a further fall in hematocrit values is evident between the second and third hours.

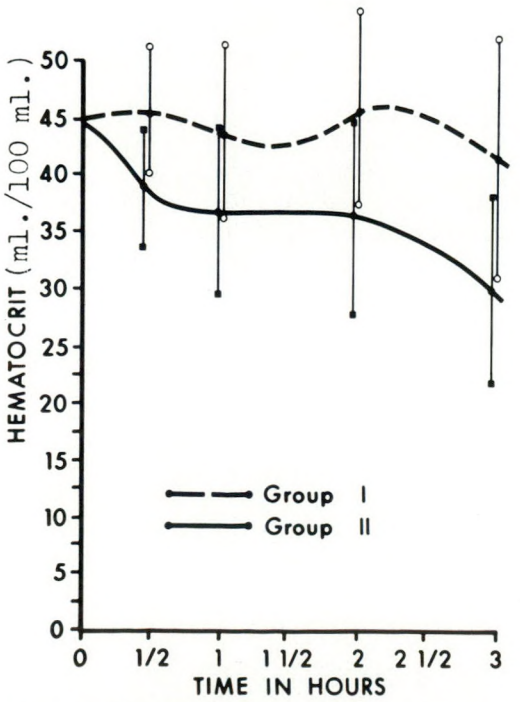

Fig. 2.—Changes in hematocrit in both groups during three hours of extracorporeal circulation.

As shown in Fig. 3 the additional amount of priming solution necessary to maintain a constant flow is much less

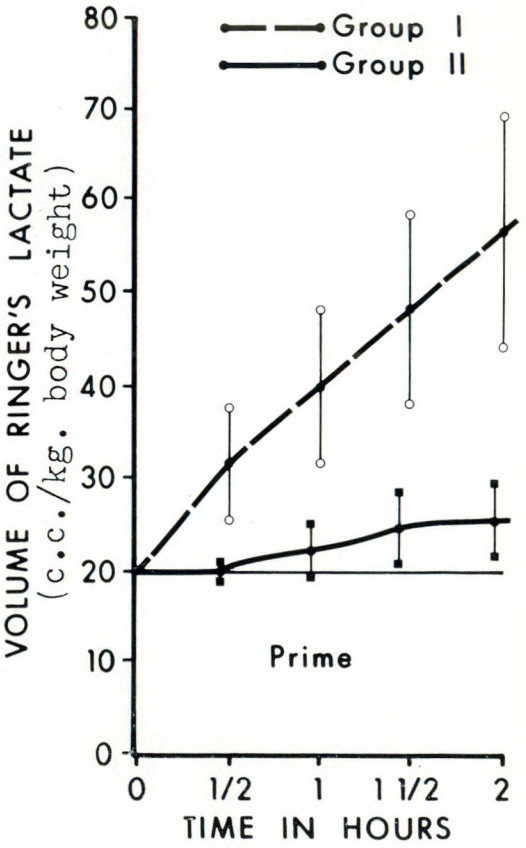

Fig. 3.—Effects of 3% glycerol on the amount of extra priming solution necessary to maintain a constant oxygenator level.

when the solution contains glycerol. At the end of three hours, more than twice the amount of priming solution had been added to the dogs in Group I as compared to those in Group II.

Discussion

Several recent reports[1, 2] on the use of frozen blood have shown that human erythrocytes can be stored in a glycerol solution at temperatures below freezing for long periods of time and subsequently

successfully transfused. Earlier work by Ustimowitsch[3] and Cugusi[4] indicated that intravenous injections of glycerol in a normal solution of sodium chloride produced diuresis without untoward side effects. Deichmann[5, 6] and Sloviter and Tietze[7] have reviewed the toxicity of glycerol solutions and conclude that glycerol given intravenously is non-toxic when administered in isotonic saline solution. In addition, Moss, Reed and Riddell[8] have reported on the use of glycerol in preserving and perfusing organs such as the canine liver, again without producing toxic effects.

Glycerol (1,2,3-propanetriol), a trivalent alcohol, is metabolized through a series of intermediate stages, similar to the carbohydrate-oxidation cycle.[9] In animals, glycerol normally constitutes about 1% of their body weight.

Clinical use of glycerin (a 10% solution) as a treatment for elephantiasis was recommended in 1938[10] and was used without obvious clinical reaction. This behaviour could well be expected as it is known[5, 11, 12] that glycol is converted by the liver to glycogen and other carbohydrates.

Our present experiment was designed to determine if a solution of 3% glycerol in Ringer's lactate would minimize the trauma to erythrocytes during repeated circuits through an extracorporeal circulation, and whether or not it would accomplish this without demonstrable toxic effects on the experimental animal. Further, an attempt was made to evaluate the effect of a glycerol priming solution on the amount of additional solution necessary to maintain a constant flow during the period of cardiopulmonary bypass.

The results of this study, as shown in Fig. 1, indicate that plasma hemoglobin levels rose more sharply in the group of dogs not receiving the glycerol priming solution. In addition, the amount of priming solution required to maintain a constant level was much greater in these animals (Fig. 3). Both these results indicate that the addition of glycerol to the prime is advantageous. Hematocrit changes, as shown in Fig. 2, showed a fall in both groups, more noticeable in the group receiving glycerol. While no definite explanation can be advanced for this, autohemodilution might be implicated since glycerol increases plasmatic concentration.[9, 13]

The large standard deviation indicated in Figs. 1 to 3 is a reflection of the small sample size taken. Only with an infinite sample size is it possible to ascertain with absolute certainty whether two populations are different. Therefore we must accept that in Fig. 2 the two groups are not clearly separated, while in Fig. 1 the groups are likely separated after two hours. In Fig. 3 the groups are almost certainly separate throughout the run.

The changes in plasma hemoglobin were assumed to be due to traumatic destruction by the extracorporeal apparatus. The lower plasma hemoglobin level in the glycerolized group of dogs could be due in part to stabilization of the surface membrane of red blood corpuscles by the glycerol solution.[1] We postulate that the smaller volume of secondary priming solution required could be due to two factors: (1) 3% glycerol in Ringer's lactate solution does not escape from the intravascular to the extravascular space as rapidly as Ringer's lactate solution, or (2) although 3% glycerol in Ringer's lactate solution is approximately iso-osmotic, it may draw some fluid from the extravascular to the intravascular space. Thus the possibility of water intoxication as well as of subsequent cardiac arrhythmias is reduced.

As mentioned, two dogs were subjected to serial laboratory determinations for three days to ascertain any possible toxic effects of glycerol. During this period, hemoglobin, hematocrit, serum electrolytes and blood urea nitrogen remained stable and within normal limits.

In view of these observations we have undertaken an extended study using this priming solution. Acid-base balance and intracellular and extracellular cation changes are being monitored during and after cardiopulmonary bypass. If these results are not anomalous, clinical application may be indicated.

Summary

Three per cent glycerol in Ringer's lactate solution was used as a priming solu-

tion for extracorporeal circulation. It was found to minimize the traumatic effects of the bubble oxygenator on the erythrocytes. It was also shown to be highly advantageous owing to the smaller volumes of additional priming solution necessary to maintain adequate flow.

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Résumé

Cette expérience ayant pour but d'établir le point de savoir si une solution de lactate de Ringer à 3% de glycérine comme soluté d'amorce de la circulation extracorporelle pouvait avoir une valeur pour éviter la destruction traumatique des éléments du sang, particulièrement les érythrocytes.

Nous avons soumis 16 chiens à une dérivation cardiopulmonaire et avons utilisé à cet effet l'oxygénateur à bulles. La vitesse de perfusion et celle du courant d'oxygène ont été maintenues constantes. Nous avons amorcé l'oxygénateur à bulles (en plastique uniservice) au moyen d'une solution de lactate de Ringer donnée à concurrence de 20 ml/kg chez quatre chiens (groupe I) et du même volume de lactate de Ringer mais contenant 3% de glycérine chez les 12 autres animaux (groupe II). La valeur de l'hémoglobine plasmatique et l'hématocrite ont été calculés à intervalles préfixés chez tous les chiens.

Ces valeurs ont été plus élevées chez les chiens qui recevaient la solution de Ringer sans glycérine que chez les autres. La quantité supplémentaire de solution d'amorce qu'il a été nécessaire d'ajouter à la solution initiale a été moindre pour le groupe II que pour le groupe I.

Il s'ensuit que la solution de Ringer à 3% de glycérine a permis de minimiser les effets traumatiques de l'oxygénateur à bulles sur les érythrocytes et de diminuer le risque d'intoxication hydrique et des arythmies cardiaques qui suivent la perfusion du contenu total de l'oxygénateur.

ANGIOGRAPHIC DIAGNOSIS OF STRANGULATED BOWEL OBSTRUCTION*

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STRANGULATED bowel obstruction is a lethal condition that may defy diagnosis by all techniques short of laparotomy. Laparotomy is certainly necessary in cases of simple bowel obstruction, but there are patients with pancreatitis, patients with bowel obstruction together with serious concomitant disease, and patients who have had multiple previous laparotomies, who are better treated conservatively if strangulated bowel obstruction can be definitely excluded. The use of pneumoperitoneum recommended by Perry, Von-Drashek and Wangensteen[1] or the emptying rate of the stomach after a Gastrografin (diatrizoate methylglucamine 76%) meal, suggested by Vest,[2] have not proved acceptable in helping to make this critical diagnosis. We have explored the use of angiography in order to differentiate between simple and strangulating bowel obstruction in the laboratory animal.

MATERIALS AND METHODS

The following investigation was carried out on 39 mongrel dogs.

Preoperative superior mesenteric artery angiograms (done under anesthesia by the Seldinger technique) were obtained from the first 25 animals. This step was omitted in the subsequent experiments. A few days later at laparotomy, an 18-inch loop of ileum, with its distal point six inches above the ileocecal junction, was obstructed with an encircling ligature of umbilical tape. In 19 animals this tape was drawn tight enough to obstruct the bowel and the venous return but not the arterial flow. In seven dogs, the artery was obstructed as well as the vein and the loop of bowel, and in the remaining 13 the tape was tied so as to obstruct the bowel, but not to impair its blood supply.

Postoperative superior mesenteric angiograms were obtained at times which ranged from 1 to 24 hours after the laparotomy. The animals were then sacrificed and an autopsy was performed.

RESULTS

All the dogs with venous, and with arterial plus venous, obstruction had strangulated bowel at autopsy. In all 26 there was persistence of dye in one or more small arterioles far in excess of the normal filling phase (Fig. 1). In 25 angiograms the arterioles were easily identified, but in the remaining one they were small enough that the result was labelled equivocal, although the radiologist (R.F.C.) is sure that it was not normal. These arterioles remained filled from four to nine times the duration of the arterial phase.

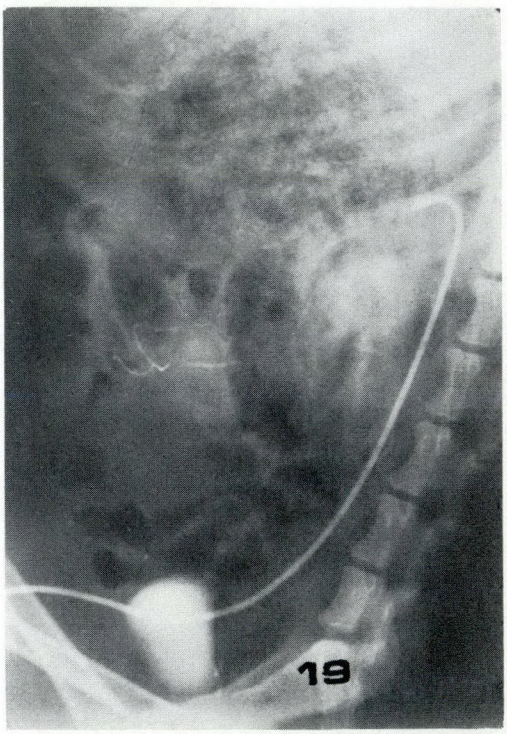

Fig. 1.—Persistent arteriolar filling, consistently found with strangulating obstruction.

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There was, in addition, spasm of the superior mesenteric artery and its branches —but neither this, nor the time of onset or duration of bowel blush, nor the characteristics of the venous phase, were of diagnostic help.

Thirteen animals were found not to have strangulated bowel at autopsy. Ten of these had normal postoperative angiograms and three had "positive" angiograms (small arteriole persistence).

Discussion

This work confirms Aakhus'[3] report of arteriolar persistence in seven animals with strangulated bowel obstruction. The explanation of this persistence of arteriolar filling has not been clearly stated. It seems likely that at the periphery of the strangulated loop there is a "transition zone" between the area of completely occluded arterioles and the normal vessels where dye enters and leaves rapidly. Fig. 2, made at four seconds, during the height of the arteriolar phase, clearly demonstrates a dilated loop with good arterial flow to each end and a "bare area" in the centre to which no blood flow is evident. Fig. 3, made in the same animal at 19 seconds, shows that the arterioles at the margins of the former "bare area" remain opacified well beyond the arterial filling phase. We believe this region constitutes a "transition zone" where arteriolar filling occurs but emptying is slow because the capillary and venous run-off is partially occluded.

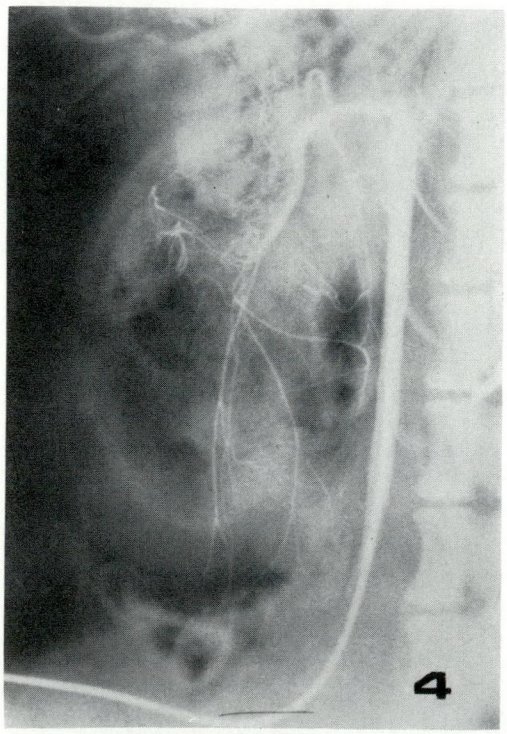

Fig. 2.—Filling phase at four seconds shows dilated (strangulated) bowel. Blood flows only to the periphery of the loop.

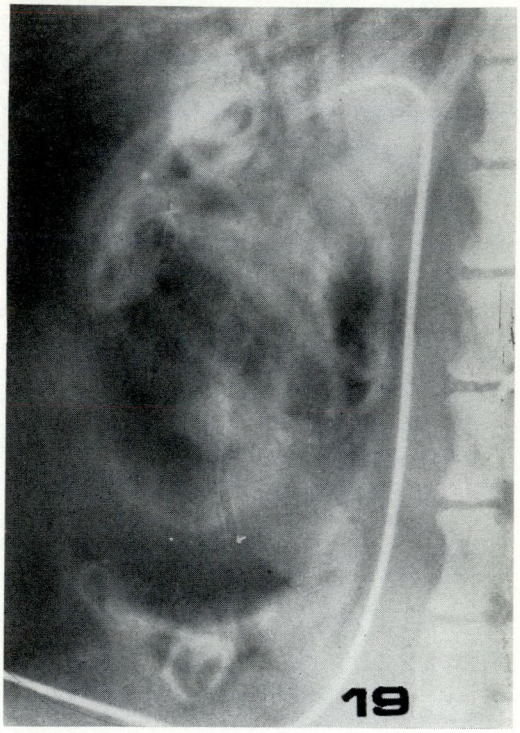

Fig. 3.—The same angiogram at 19 seconds. Arterioles at the periphery of the loop remain opacified.

The same explanation may apply to the "false positives" found in the animals with obstruction but no gross strangulation. Most of these angiograms were performed about eight hours after obstruction and it may be that the arteriolar stasis demonstrated would have progressed to complete obstruction if more time had been allowed. This could be a very early gross sign of impending strangulation of bowel.

These findings are preliminary. If clinical trials bear out the laboratory findings, "false positives" may occur in some patients before strangulation is apparent. This error might lead to surgical exploration before it is mandatory—a much less

serious error than delay in the patient with actual strangulated bowel obstruction. In the laboratory there were no "false negatives" among the 26 animals with strangulating obstruction, so we may anticipate that this error will occur rarely if at all.

We have done superior mesenteric angiograms on six patients with bowel obstruction. All were "negative". Four patients were found to have viable bowel at operation and two recovered without laparotomy.

Summary

In the laboratory superior mesenteric angiograms have made possible correct identification of strangulated bowel in 26 dogs. The result of only one of these tests was equivocal. In 13 dogs with obstruction that had not progressed to strangulation there were three "false positives".

It is hoped that angiography will help in the diagnosis of strangulation in patients with bowel obstruction in whom operation otherwise might be deferred.

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Résumé

Il importe de faire rapidement le diagnostic d'occlusion intestinale par étranglement, car cette pathologie exige une laparotomie immédiate; par contre, il est généralement préférable de traiter par des moyens conservateurs l'occlusion intestinale simple en attendant que l'état du malade soit meilleur. Nous avons étudié la méthode angiographique, dans les conditions contrôlées du laboratoire, pour identifier l'apport sanguin compromis qui accompagne l'étranglement.

Les expériences ont été faites sur 39 chiens. Quelques jours après avoir pris des artériogrammes contrôlés du mésentère supérieur, on a procédé à la laparotomie, moment auquel on a procédé à l'occlusion d'une anse standard de 18 pouces d'iléum, au moyen d'un ruban ombilical serré assez fort pour entraver la circulation. Des angiogrammes ont été pris ensuite dans un délai variant d'une heure à 24 heures après l'opération.

On a constaté une anomalie caractéristique de l'angiogramme, débutant deux heures après l'occlusion et persistant pendant 24 heures. Cette anomalie a été constatée chez 25 chiens qui avaient une gangrène intestinale à l'autopsie. Un seul angiogramme n'a pas permis de poser de diagnostic. Tout aussi éloquent est le fait que ce signe n'a pas été découvert chez les 13 animaux qui n'avaient pas de gangrène intestinale.

Dans les cas expérimentaux d'occlusion par étranglement, les angiogrammes ont mis en évidence la persistance du remplissage des petites artères longtemps après la disparition du courant artériel du reste de l'intestin. Nous espérons que cette anomalie facilement reconnaissable facilitera chez l'homme le diagnostic de l'occlusion intestinale par étranglement.

CATHETER VERTEBRAL ANGIOGRAPHY

Direct catheter vertebral artery angiograms were obtained via the femoral and axillary routes in 300 patients. This technique is valuable in the diagnosis of expanding lesions in the posterior fossa and is particularly valuable in the recognition of vascular lesions.

Neurologic complications were encountered in only 10 of the 300 selected vertebral artery angiograms. They included transient hallucination and mild disorientation. In two patients mild hemiparesis developed, but disappeared within 48 hours. Two long-lasting complications developed; one of these was a quadriplegia after the injection of contrast material into the thyrocervical trunk with the subsequent development of transverse myelitis. The other long-term complication was the development of an occlusion in the distal portion of the left vertebral artery, most likely due to intimal damage by the catheter. Vertigo and dysphagia developed along with a left facial palsy and ptosis. The symptoms and signs cleared almost completely in the ensuing five months.

Posterior fossa tumours and vascular lesions and even supratentorial tumours were diagnosed with considerable accuracy. The rate of technically successful vertebral angiograms in this series was 95%.—Takahashi, M., Wilson, G. and Hanafee, W.: Catheter vertebral angiography; a review of 300 examinations, *J. Neurosurg.*, **30:** 722, 1969.

BOOK REVIEWERS, 1969

The physicians, other medical scientists and members of allied health professions, who month by month assist the Editor of *The Canadian Journal of Surgery* by contributing book reviews, make a significant contribution to continuing medical education. The Editorial Board and the Editors wish at this time to acknowledge, with gratitude, the assistance during the year 1969 of their colleagues whose names appear below.

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BOOK REVIEWS

ACHALASIA OF THE ESOPHAGUS. F. Henry Ellis, Jr. and Arthur M. Olsen. Vol. 9 in the series Major Problems in Clinical Surgery. 221 pp. Illust. W. B. Saunders Company, Philadelphia; W. B. Saunders Company Canada Limited, Toronto, 1969. $9.75.

This is an excellent monograph on the subject by two experts in the field. There is a good historical account of the condition and of the individuals who have been active in its study and treatment. Current and past ideas on pathogenesis are reviewed and the reader is left with the suggestion that it is possibly the result of disease of the motor nucleus of the vagus nerve. The physiology of deglutition is described and the modern pressure-study techniques are well explained. The disordered physiology in achalasia is well discussed, and the use of these pressure studies in diagnosis is described.

The clinical aspects of the disease are clearly recorded. A good historical review of previous methods of treatment is given and then the modern methods are discussed. The two authors give a report of the extensive experience at the Mayo Clinic over 63 years. Up to 1936, 948 patients, and from 1936 to 1967 a further 1416 patients, were treated by dilatation. From 1949 to 1969, 300 esophagomyotomy operations were performed. The authors now strongly advocate surgical treatment of the condition rather than forcible dilatation. They point out, however, that many other clinicians recommend operation only if a trial of forcible dilatation is unsuccessful. The reviewer would strongly agree with this latter opinion.

The references are extensive, the illustrations are good, and the general format of the book is excellent. For a long time this work will remain an authoritative report on achalasia of the esophagus.

ATLAS OF GASTROINTESTINAL SURGERY. Komei Nakayama. 649 pp. Illust. J. B. Lippincott Company, Philadelphia; J. B. Lippincott Company of Canada Ltd., Toronto, 1968. $46.00.

This volume of 650 pages is a huge *tour de force* on the surgical attitudes and ideas of one of the eminent surgeons of Japan. It presents the ideas of Dr. Nakayama on a variety of operations which range over the entire field of gastrointestinal surgery, usually considered the domain of the general surgeon. It is a reasonably intimate look at one man's views in relation to a series of operative procedures which he obviously performs with considerable skill, dexterity and thoroughness. On the right-hand pages are series of drawings to illustrate the surgical conditions, concepts, operative steps and results of the operations. The left-hand pages contain a discourse on the indications, technique and instrumentation as well as brief notes about the peculiar and special portions of the operative procedure under description.

This is an exhaustive presentation of one man's attitudes and ideas. The illustrations are exquisitely simple in the demonstration of some procedures, yet are poorly done in the depiction of some of the complex anatomy seen within the abdomen. It is an adequate reference work which is exhaustive in the wide scope of the operative procedures described but not in the depth of any single description. It is recommended for every residency program. It probably is of less value on the shelf of a talented and well-trained surgeon.

BASIC SURGICAL PHYSIOLOGY. Edited by Frederick W. Preston and John M. Beal. 508 pp. Illust. Year Book Medical Publishers, Inc., Chicago, 1969. $27.50.

As can be expected in a volume produced by the staff of a single department, considerable variation in the approach adopted by the various authors is encountered. The general standard of the chapters is high and some are absolutely outstanding.

The volume is laid out in seven sections corresponding to major systems, each section containing a number of chapters. The first section, entitled "General Considerations", contains an excellent chapter on wound healing, short and somewhat variable chapters on the other basic subjects. The chapters on hemorrhagic shock and shock associated with infection are somewhat disorganized and refer mainly to old references in the respective bibliographies. The treatment of the biological principles of transplantation is much more successful.

The circulatory system is well dealt with, particularly the chapters on blood and blood coagulation which constitute excellent reviews of these subjects. By far the most successful section is that on the respiratory system which is carefully done, modern in context and most valuable to student and practitioner alike. The section on the digestive system is satisfactory, except perhaps for the chapter on the stomach which for some reason omits consideration of gastric secretory tests and their relationships in peptic ulcer disease. The chapters on the pancreas are outstanding. The physiology of the endocrine glands is handled in a thorough and helpful fashion, especially the chapter on the parathyroid which is very up to date and interesting.

The volume fills a needed space in the surgical literature at the present time and on balance can be strongly recommended.

BIOPSY PROCEDURES IN CLINICAL MEDICINE. Edited by A. E. Read. 193 pp. Illust. John Wright & Sons Ltd., Bristol; The Macmillan Company of Canada Limited, Toronto, 1968. $6.50.

This small text is remarkably useful and easy to read. It can be recommended to any intern, resident or practitioner. Biopsy procedures on almost every organ in the body are described and from sites that are not usually considered when biopsy is thought of, e.g. lymph glands, spleen and gastrointestinal tract.

What the reviewer found most useful was the accurate detail in which the techniques are described so that they can be carried out by someone who is inexperienced in the procedures. In many texts and journals these details are not included but only general directions such as "use a needle of adequate size" or "choose an appropriate site for the puncture" are given. In addition, this text states clearly the indications and contraindications for biopsy, together with the dangers and complications.

COLLATERAL CIRCULATION IN CLINICAL SURGERY. Edited by D. E. Strandness, Jr. 633 pp. Illust. W. B. Saunders Company, Philadelphia; W. B. Saunders Company Canada Limited, Toronto, 1969. $20.00.

The authors hope that this book will be useful to the experienced physician, vascular surgeon, surgical specialist and fledgling house officer, and certainly the publication should fulfil such hopes. The text covers collateral circulation in those areas of the body that are of great significance to surgeon and physician alike in understanding disease and in treating it intelligently. Naturally those areas that lend themselves to constructive therapy such as the ischemic limb and obliterative vascular disease of the cerebral and coronary artery vessels receive appropriately full description.

It seems a pity that the authors did not seize the opportunity to enlarge on present knowledge and concepts of the importance of the microcirculation in terms of collateral supply, its reaction to injury and disease and its response to medical and surgical therapy. The ultimate collateral circulation which keeps the tissues alive is the microcirculation and failure to stress its importance has played a great part in our tardy understanding of many clinical entities such as medical or surgical shock.

The section on collateral circulation of the heart is particularly well done and here one gets into the mystery and excitement of those small vessels which keep us alive. However, in spite of the 1969 publication date, no mention has been made of the work of Baird on internal mammary artery implantation and his theory of collateral development thereafter.

The volume is clearly and cleanly printed, with ample illustrations and diagrams. It is a praiseworthy initial presentation of an interesting and important aspect of the acquisition and application of medical knowledge and science. New knowledge will mean review and modification of the text and it is to be hoped that one can look forward to future editions of this book as and when such new information calls for them.

CONTROVERSY IN OBSTETRICS AND GYNECOLOGY. Edited by Duncan E. Reid and T. C. Barton. 414 pp. Illust. W. B. Saunders Company, Philadelphia; W. B. Saunders Company Canada Limited, Toronto, 1969. $16.75.

The editors of this work have had the novel idea of assigning the various areas of their specialty about which there is disagreement to advocates of opposing views. After the evidence for the differing opinions has been offered, the editors summarize the arguments and attempt an adjudication. The pleasure with which one approaches a work that promises so much is tempered with surprise that in what is commonly believed to be such an orderly field within the practice of medicine, so much room for controversy should exist to justify such a large volume. Chapter headings mentioning ectopic pregnancy, breech delivery and resuscitation of the newborn partly explain its size, for one does not usually consider that these topics evoke much serious disagreement among obstetricians. Attention to the text reveals that much of the material included is by no means controversial. Some of the contributors have taken advantage of the opportunity to set forth their views at great length and to discuss the whole subject, not merely its controversial aspects. This means that often the actual points on which there can be important differences of opinion and on which enlightenment is sought, are obscured by too prolix a presentation. In some cases authors assigned the same subject actually deal with different problems, so that the points of view are not strictly alternatives. Elsewhere, however, the requirements of the title are satisfied as, for example, in the discussion of premature rupture of the membranes.

The book contains much of great practical value and can be read with profit. Had the editors exercised stricter control over their contributors its impact would have been greater, for the underlying idea is an excellent one and deserves wider application.

EDWARD STEVENS. GASTRIC PHYSIOLOGIST, PHYSICIAN AND AMERICAN STATESMAN. Edited by Stacey B. Day. 179 pp. Cultural and Educational Productions, Cincinnati and Montreal, 1969. Price not stated.

The editor of this small volume presents the reader with a thoroughly researched series of papers relating to the life of Dr. Edward Stevens who was born in or about 1755 in the West Indies and received his medical education at the University of Edinburgh. During

this time, he had a primary interest in gastric digestion on which he gave an inaugural dissertation in 1777. The translation of this work from the original Latin is the highlight of the volume and gives the reader and student of gastric physiology an insight into the thought and experiments (conducted in humans as well as animals) of two centuries ago. It is apparent that, although forgotten, the work presented must have had a major effect on students of gastric secretion during this period. Dr. Day has given us a first-hand glimpse of this historical document.

The book deals also with the letters that have been unearthed which passed between Edward Stevens and Benjamin Rush during the yellow fever epidemic in Philadelphia in 1793. In this interlude, Edward Stevens is linked with other American statesmen and may have had some influence on the origins of medical teaching in the United States at the turn of the eighteenth century. There is an account of the part Dr. Stevens appeared to have played as consul-general of the United States in Santo Domingo, illustrated by diverse communications written during this third part of his life.

This book serves to illustrate the contributions of a hitherto unknown physician, Edward Stevens, in the fields of gastric digestion and American history and represents a historical study of this era. Although a credit to the industry of the editor, it is not a very readable book and will attract only students of the history of medicine.

HARVEY CUSHING. Selected Papers on Neurosurgery. Edited by Donald D. Matson, William J. German and a Committee of the American Association of Neurological Surgeons. 669 pp. Illust. Yale University Press, New Haven and London; McGill University Press, Montreal, 1969. $19.25.

To mark the centennial of Harvey Cushing's birth, the American Association of Neurological Surgeons (founded as the Harvey Cushing Society) has sponsored the publication of this book which contains many superb papers on neurosurgical topics by Cushing and a complete bibliography of his works. Although the papers are all available in the various journals in which they were published during his lifetime, it is an advantage for the present generation of students and practitioners of neurosurgery to have them collected under one cover.

The book is divided into seven chapters, the titles of which show the great scope and breadth of Cushing's endeavour: "Trauma to the Nervous System"; "Trigeminal Neuralgia"; "Blood Pressure"; "Neoplasms"; "Cerebrospinal Fluid"; "Pituitary and Hypothalamus"; and "Essays". The papers contain many facts which are helpful in the practice of neurosurgery today. But, equally important as "the facts" is the method, and Cushing's genius was the application of the scientific method to clinical problems. This is well demonstrated in the paper "Peptic Ulcers and the Interbrain" in which he recounts how he was stimulated to examine experimentally the neurogenic theory of peptic ulceration when peptic ulcers occurred in several of his patients.

In "Essays" we are treated to Cushing's thoughts on super-specialization. His "Homo Chirurgious" is a satirical admonition to all surgeons to maintain an interest and knowledge in fields of medicine other than their own. Also included is the address given at his seventieth birthday party where this idea was succinctly expressed, ". . . practically all disorders, to which mind and body are heir, first or last come to affect the nervous system".

In essence, the book is a superb demonstration of the scientific method in clinical medicine applied by a mind with scope unlimited by artificial boundaries. The correctness of his philosophy of medicine is proved by the relevance of the book to the practice of medicine today.

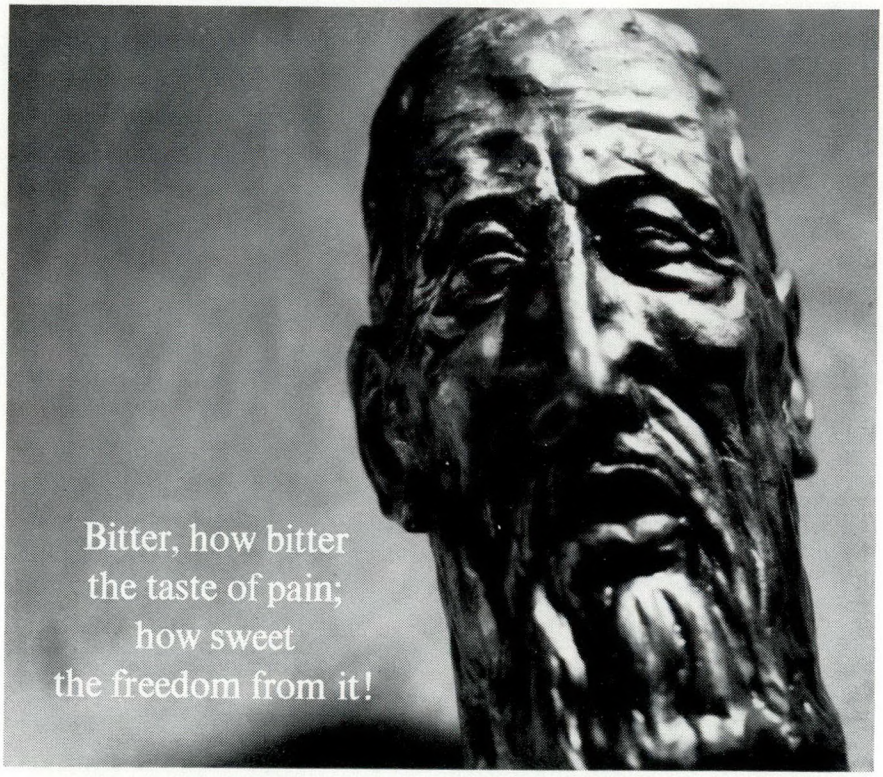
ILLUSTRATED HANDBOOK IN LOCAL ANAESTHESIA. Edited by Ejnar Eriksson. 160 pp. Illust. Year Book Medical Publishers, Inc., Chicago, 1969. $14.25.

This new book on local anesthesia techniques is well edited. The photography and drawings are of the highest quality and take advantage of the most modern techniques and skills. The editor and his technical staff are to be praised for having achieved what they set out to accomplish: a simple and clear presentation of the most common and useful nerve blocks. Reading this book is entertaining and useful, because the material can be followed easily and put into practice accurately. However, there is a great deal of oversimplification and too many of the problems connected with conduction anesthesia are approached in a superficial manner, while the pharmacology is practically non-existent. In 1969 any book should, in the reviewer's opinion, be much more specific and at least offer some recent and accurate references. In conclusion, while the editor is to be commended for a book which is as easy to read as a magazine, attention should be drawn to the fact that it is not adequate for preparing a candidate for examination. Its value is limited to refreshing the physician's memory of a nerve block that he may not have done for some time. The price, on the other hand, is not easily forgotten.

THE LIVER. Colston Paper No. 19. Edited by A. E. Read. 405 pp. Illust. Butterworth & Co. (Publishers) Ltd., London; Butterworth & Co. (Canada) Ltd., Toronto, 1967. $31.50.

These proceedings of a Symposium held at the University of Bristol in April 1967 represent a useful addition to the library shelf.

(Continued on page 100)



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Approximately 40 papers are included. The authors are gathered from both sides of the Atlantic and the majority are internationally known. Most exhibit a close personal involvement with the work they describe. Outstanding contributions are provided by A. J. Levi on drug hepatitis, by H. Popper on acute hepatic necrosis in hepatitis, by R. Williams, L. Kreel and L. M. Blendis on celiac axis catheterization, by S. Sherlock on neuropsychiatric changes following portasystemic shunting and by J. Terblanche and A. G. Riddell on the strategy of liver transplantation. The selection of papers is somewhat unusual for a general symposium in that great weight is given to the problem of portal hypertension and liver transplantation. This emphasis adds a great deal of interest for the surgical reader. W. V. McDermott's discussion of colonic operations for hepatic encephalopathy is the only major disappointment since the published paper appears to be little more than an abstract.

One hesitates to suggest that the book is worth the price, which no doubt reflects the glossy printing, but it is certainly worth borrowing for a night or two.

LA MAIN RHUMATOIDE. The Rheumatoid Hand (in French and English). Under the direction of Raoul Tubiana. 269 pp. Illust. Expansion Scientifique Française, Paris, 1969. Price not stated. Paperbound.

This monograph is a "second edition" of a collection of papers on the rheumatoid hand. It includes all those presentations of the Group d'Etude de la Main (G.E.M.) session of March 1966, which appeared in the first edition, together with those of other meetings that have taken place since that time. All articles are in both French and English. The authors are recognized authorities in the field for discussion.

This edition has been improved by the grouping of the articles into chapters. Each chapter emphasizes certain features of the disease or its deformities. An excellent review of bone and joint anatomy and fractures leading to ulnar deviation of the fingers is presented in chapter 1. Two chapters are taken for the discussion of the medical management, non-operative treatment, splinting and rehabilitation of the patient with rheumatoid disease. The remaining six chapters are primarily papers on the surgical treatment. The surgery of the joints in the hand and wrist includes discussions on arthroplasty, fusion, silastic and metal prostheses, and synovectomy. The types and management of tendon lesions and the deformity resulting from imbalance of intrinsic and extrinsic musculature are presented. There is a good chapter on the deformities occurring in the thumb. A most interesting chapter includes the round-table discussion on the rheumatoid hand that took place at the Anglo-Scandinavian Symposium on Surgery of the Hand held in May 1967.

The potential of surgery to prevent and correct rheumatoid deformities of the hand has been widely appreciated for only the past decade. No surgeon or clinic has been able to have an adequate follow-up of a significant number of patients to assess the results of many of the procedures used for this condition. The topic, therefore, is ideally suited for this type of monograph. The collecting and editing of the papers has been commendably done.

This monograph can be regarded as required reading for surgeons interested in the rapidly evolving specialty of hand surgery, for rheumatologists who are constantly encountering patients with hand disabilities, and physiatrists concerned with splinting and rehabilitation.

LA MEDIASTINOSCOPIE. R. Sarrazin and R. Voog. 104 pp. Illust. Masson et Cie, Paris, 1968. 21 F. $4.65 (approx.).

One must agree that the surgeon is best armed who is best informed concerning the nature and extent of the disease for which he is preparing to operate. Therefore, without putting too fine a point upon it, one could likewise agree that mediastinoscopy is a useful diagnostic tool. For various reasons the procedure has never become as popular on the American continent as it has in Europe and relatively fewer physicians on this side of the ocean have advocated or become proficient in its use. This authoritative little book of scarcely 100 pages, with its precision and clarity, could therefore well serve to extend the acceptance of the procedure in our diagnostic armamentarium.

All important aspects of the subject are treated in detail and the material is admirably organized. It begins with a historical précis, followed in order by anatomic considerations, the operative procedure itself (including anesthesia and a description of instruments and materials), indications and contraindications, a section entitled "Incidents and Accidents", which describes possible pitfalls, and a clinical study. The latter describes the authors' experience with 150 cases of bronchial cancer, sarcoidosis, tuberculosis, silicosis, mediastinal tumours and other, rarer, conditions. Since mediastinoscopy in the hands of many physicians has been associated with higher morbidity than is reported in the present series, the step-by-step detailed description of the operative technique together with the nicely illustrative diagrams and photographs of actual anatomic preparations are particularly interesting and valuable.

This useful book clearly deserves to be read by all students of the thorax and therefore merits translation for those who cannot read French.

(Continued on page 102)

(Continued from page 100)

THE SPINE. A Radiological Text and Atlas. 3rd ed. Bernard S. Epstein. 730 pp. Illust. Lea & Febiger, Philadelphia; The Macmillan Company of Canada Limited, Toronto, 1969. $38.50.

This third edition of Dr. Epstein's book will find a valued place on the bookshelf of the orthopedist, neurosurgeon and radiologist. Once more, the range of material presented has been increased. Radiological anatomy is covered precisely and with great clarity, both in text and illustrations. Many special techniques employed in the investigation of the spine are described. In over 700 well-filled pages the author discusses the spine, from malformations and metabolic disease to injuries and degeneration.

Understandably, even in a book of this length coverage of certain, especially rare, conditions has to be curtailed. None the less, to find only seven pages of text, including three radiographs, on scoliosis is a disappointment. No attempt is made to describe the radiological details, particularly the differences that occur among idiopathic, paralytic, congenital and neurofibromatosis scoliosis.

With modern techniques of printing and x-ray reproduction, little need be lost in clarity of roentgenogram illustrations. Unfortunately, this does not hold true for this book. Much of the value of this large compendium of material is dissipated due to poor reproduction. In some illustrations (e.g. Figs. 62D, 75E, 87B and 190A & B) the region is hardly recognizable.

These limited criticisms aside, this book probably represents the largest and most detailed coverage of the spine that exists in a single volume.

SYNDESMOLOGY OR A DESCRIPTION OF THE LIGAMENTS OF THE HUMAN BODY. Josias Weitbrecht. Translated by Emanuel B. Kaplan. 197 pp. Illust. W. B. Saunders Company, Philadelphia; W. B. Saunders Company Canada Limited, Toronto, 1969. $32.40.

Both the translator and publisher are to be heartily congratulated on this most excellent production of Weitbrecht's "Syndesmologia". This is, unfortunately, a little-known work but one which played a major role in the evolution of human gross anatomy. It should be in every anatomy library if only to show the new generation of anatomists that anatomy began before the invention of the electron microscope.

Josias Weitbrecht was born at Schorndorf, Germany, in 1702 and in 1725 became one of the many distinguished foreign scientists who worked at the St. Petersburg Academy of Sciences in Russia. He began as a prosector in the Institute of Comparative Anatomy, and was made professor of anatomy and physiology in 1731, a post he held until his sudden death at the age of 45 years. The famous anatomical collection of the Dutch anatomist, Ruysch, was one of the priceless treasures that Peter the Great, Tsar of Russia, purchased for his newly created Academy of Sciences. One of Weitbrecht's early achievements was the "Compendium Anatomicum" of 1729 which described completely this remarkable collection.

Weitbrecht spent many years personally dissecting fresh cadavers to obtain the specimens for his comprehensive "Syndesmologia" but, as he frankly admits, he did not ignore the work of others. In particular, he acknowledged his debt to the careful descriptions in the "Expositiones Anatomicae" of Winslow. Although Weitbrecht had completed much of his work when Winslow's work appeared he repeated and re-explored his own observations. He summed up his feelings in a single short sentence: "I am grateful for the use of this illustrious man's work."

One sentence in Weitbrecht's introduction admirably described his attitude as an author: "I wrote clearly and factually, so as not to effect a sublime style, but at the same time not to oversimplify." Nowhere is this style better seen than in the first section entitled "Of Ligaments in General". His definition of what is or is not a ligament is far more lucid than most current textbooks of anatomy. The illustrations are excellent and great praise should be given to his illustrator, Andreas Grecow and his engravers, Gregorius Katchalow and Johanes Sokalow.

Jessie Dobson's compilation of anatomical eponyms attaches his name to three structures: the foramen ovale in the capsule of the shoulder joint, the retinacular fibres of the femoral neck and the oblique radioulnar ligament. Whilst all present-day anatomists know the foramen attributed to his contemporary, Winslow, few, if any, know the name of Weitbrecht. Perhaps this may be due to the miserable history of the previous translations of the "Syndesmologia". A French version, by M. Tarin, was published in 1752 but reduced a quarto of 278 pages to an octavo of 144 pages. The German version was taken from the French and the English version, by Robert Harrison (1829), was a mere 62 pages. All interested in the history of human anatomy owe a great debt to Dr. Emanuel B. Kaplan for this first complete translation of this monumental work.

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Books Received

Books are acknowledged as received, but in some cases reviews will also be made in later issues.

The Application of Radioiodinated Rose Bengal and Colloidal Radiogold in the Detection of Hepatobiliary Disease. Leonard Rosenthall. 88 pp. Illust. Warren H. Green, Inc., St. Louis, 1969. $7.50.

Curiosities of Medical Experience. 2nd ed. J. G. Millingen. 566 pp. Singing Tree Press, a division of Gale Research Company, Detroit, 1969. $16.50.

Diseases of the Chest. 3rd ed. H. Corwin Hinshaw. 799 pp. Illust. W. B. Saunders Company, Philadelphia; W. B. Saunders Company Canada Limited, Toronto, 1969. $27.00.

Experience in Hepatic Transplantation. Thomas E. Starzl with the assistance of Charles W. Putnam. 553 pp. Illust. W. B. Saunders Company, Philadelphia; W. B. Saunders Company Canada Limited, Toronto, 1969. $40.50.

Manual of Surgical Therapeutics. Edited by Robert E. Condon and Lloyd M. Nyhus. 379 pp. Little, Brown and Company, Boston; J. B. Lippincott Company of Canada Ltd., Toronto, 1969. $6.45. Paperbound.

Microneurosurgery. Robert W. Rand. 224 pp. Illust. The C. V. Mosby Company, St. Louis, 1969. $27.50.

Nouveau Traité de Technique Chirurgicale. Tome XI. Intestin Grêle–Côlon–Rectum–Anus. Jean Lamy and others. 682 pp. Illust. Masson et Cie, Paris, 1969. 180 F. $40.00 (approx.).

Nouveau Traité de Technique Chirurgicale. Tome XIV. Gynécologie. Henry G. Robert. 820 pp. Illust. Masson et Cie, Paris, 1969. 233 F. $49.00 (approx.).

The Operations of Surgery. Vol. 2. The Upper Respiratory and Alimentary Tracts; The Thyroid and Parathyroid Glands; Chest Surgery; Management of Head Injuries; Orthopaedics; Spinal Injuries; Vascular Surgery. A. J. Gardham and D. R. Davies. 636 pp. Illust. J. & A. Churchill Ltd., London; Longmans Canada Limited, Toronto, 1969. $32.30.

Practical Management of the Allergic Child. Vincent J. Fontana. 371 pp. Illust. Appleton-Century-Crofts, Educational Division, Meredith Corporation, New York, 1969. $15.00.

Pye's Surgical Handicraft. 19th ed. Edited by James Kyle. 820 pp. Illust. John Wright & Sons Ltd., Bristol; The Macmillan Company of Canada Limited, Toronto, 1969. $10.50.

Recent Advances in Paediatric Surgery. 2nd ed. Edited by A. W. Wilkinson. 280 pp. Illust. J. & A. Churchill Ltd., London; Longmans Canada Limited, Toronto, 1969. $13.30.

Recent Advances in Surgery. 7th ed. Edited by Selwyn Taylor. 659 pp. Illust. J. & A. Churchill Ltd., London; Longmans Canada Limited, Toronto, 1969. $17.10.

Selective Arteriography of the Spinal Cord. John L. Doppman, Giovanni di Chiro and Ayub K. Ommaya. 157 pp. Illust. Warren H. Green, Inc., St. Louis, 1969. $12.50.

Shoulder Lesions. 3rd ed. H. F. Moseley. 318 pp. Illust. E. & S. Livingstone Ltd., Edinburgh and London; The Macmillan Company of Canada Limited, Toronto, 1969. $30.50.

Spezielle Chirurgie für die Praxis. Vol. 2, part 1. Edited by Franz Baumgartl, Karl Kremer and Hans Wilhelm Schreiber. 744 pp. Illust. Intercontinental Medical Book Corp., New York; Georg Thieme Verlag, Stuttgart, West Germany, 1969. DM 270,00. $73.00 (approx.).

Surgery of Acquired Vascular Disorders. Benjamin B. Jackson. 479 pp. Illust. Charles C Thomas, Publisher, Springfield, Ill.; The Ryerson Press, Toronto, 1969. $27.00.

Surgery of the Stomach and Duodenum. 2nd ed. Edited by Henry N. Harkins and Lloyd M. Nyhus. 934 pp. Illust. Little, Brown and Company, Boston; J. B. Lippincott Company of Canada Ltd., Toronto, 1969. $41.50.

Die Therapie der Koxarthrose. Edited by August Rütt. 310 pp. Illust. Intercontinental Medical Book Corp., New York; Georg Thieme Verlag, Stuttgart, Germany, 1969. DM 78,00. $11.20 (approx.).

Urologic Surgery. Edited by James F. Glenn and William H. Boyce. 770 pp. Illust. Hoeber Medical Division, Harper & Row, Publishers, Inc., New York, 1969. $32.50.

Urologische Operationslehre. Vol. 1. Various contributors. Edited by G. H. Heise and E. Hienzsch. 147 pp. Illust. VEB Georg Thieme, Leipzig, 1969. DM 46,00. $12.50 (approx.). Paperbound.

Urologische Operationslehre. Vol. 2. Various contributors. Edited by G. H. Heise and E. Hienzsch. 139 pp. Illust. VEB Georg Thieme, Leipzig, 1969. DM 44,00. $12.00 (approx.). Paperbound.

Urologische Operationslehre. Vol. 3. Various contributors. Edited by G. H. Heise and E. Hienzsch. 76 pp. Illust. VEB Georg Thieme, Leipzig, 1969. DM 24,00. $6.50 (approx.). Paperbound.